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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

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NEUROGENIC TUMORS OF THE MEDIASTINUM: A CLINICOPATHOLOGIC STUDY*

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ROCHESTER, MINNESOTA

NEUROGENIC tumors are the most common group of intrathoracic extrapulmonary tumors. In 1951, Ackerman and Taylor[1] reported on 48 proved cases of such tumors and found an additional 56 cases in the literature. In 1953, Harrington[3] reported a series of 212 intrathoracic extrapulmonary tumors of which 58, or approximately a quarter, were neurogenic.

Neurogenic neoplasms may be divided into two main groups depending on their origin: those that arise from nerve sheaths and those that arise from nerve cells. The nerve-sheath tumors, generally referred to as schwannomas (or neurilemmomas) comprise the largest group. They are thought to arise from completely differentiated adult Schwann cells, and the vast majority are benign. The nerve cell tumors are considered to be true embryonic tumors and represent a proliferation of ganglion cells from undifferentiated elements that have migrated from the neural crest to form the components of the paraganglionic autonomic nervous system. These tumors have been considered to be part of a continuous spectrum: at one end the completely undifferentiated malignant neuroblastoma and at the other end the mature, benign ganglioneuroma. Finally, there exists a small group of mediastinal neurogenic tumors, commonly referred to as "neurofibromas," that contain all the nerve elements, namely axons, sheath cells and connective tissue. A few of the neurofibromas appear grossly as a tangled plexiform mass, and when in this form they are invariably associated with the stigmata of generalized neurofibromatosis.

MATERIAL AND METHOD

The present paper deals with a study of 140 cases of extrapleural mediastinal tumors of neurogenic origin treated surgically at the Mayo Clinic over a period of thirty-three years, from 1923 through 1955 (Tables I and II).

The case records and some of the roentgenograms were reviewed. The gross specimens were examined. Sections were re-

* Presented at the Sixtieth Annual Meeting of the American Roentgen Ray Society, Cincinnati, Ohio, September 22–25, 1959.
† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

TABLE I

THE NUMBER, THE AGE AND THE SEX OF PATIENTS WITH THE VARIOUS TYPES OF NEUROGENIC MEDIASTINAL TUMORS IN OUR SERIES*

	Schwannoma	Malignant Schwannoma	Ganglio-neuroma	Neuro-blastoma	Plexiform Neurofibroma	Nonplexiform Neurofibroma	Total
Total No. of Patients	86	1	39	5	5	4	140
Sex							
Male	41	1	20	3	4	3	72
Female	45		19	2	1	1	68
Age, yr.							
0–9			10	3	3		16
10–19	5	1	10	2			18
20–29	19		4				23
30–39	22		7			2	31
40–49	20		3			1	24
50–59	13		5		1	1	20
60–69	7				1		8

* Eight of the patients had tumors of the so-called dumbbell type; 6 of these were schwannomas and 2 were ganglioneuromas. Three schwannomas arose from unusual sites, namely, the left vagus, right vagus and left recurrent laryngeal nerve.

moved from each tumor for microscopic study. At least four sections were removed at different sites from tumors of sympathetic origin.

Follow-up study in most instances was conducted by correspondence.

CLINICOPATHOLOGIC FEATURES

The presenting complaints of patients with neurogenic tumors were mainly the result of pressure on neighboring structures (Table III). In more than half of the cases the schwannomas and ganglioneuromas were asymptomatic, and the tumor was first recognized from a routine roentgenogram of the thorax. In those patients with symptoms the most common complaint was pain in the thorax, variously described as "vague," "intermittent" or merely as a "discomfort." However, in a few cases it was recorded as "root pain" by the examining physician.

TABLE II

LOCATION OF NEUROGENIC MEDIASTINAL TUMORS*

	No. of Cases
Superior mediastinum	46
Superior and middle mediastinum	15
Middle mediastinum	48
Middle and inferior mediastinum	6
Inferior mediastinum	25
Total	140

* Located by arbitrarily dividing the thorax into three equal transverse zones.

Tumors situated in the superior mediastinum when causing symptoms revealed their presence in several ways. In 2 cases pressure on the brachial plexus resulted in pain, paresthesia and weakness of the upper extremity. In 3 cases the proximity of the tumor to the stellate ganglion produced Horner's syndrome. Cough, sputum, repeated respiratory infections, shortness of breath on exertion and cyanosis were encountered as presenting complaints singly or in combination with one another and were usually associated with large tumors which had undoubtedly compressed pulmonary parenchyma to the point of interfering with respiratory function and drainage. Encroachment on, and displacement of, mediastinal structures also contributed to the symptoms. Three patients showed distention of the veins of the neck as a re-

TABLE III

SYMPTOMS AND PHYSICAL FINDINGS IN CASES OF NEUROGENIC MEDIASTINAL TUMORS

	No. of Cases		No. of Cases
No symptoms	87	Hemoptysis	2
Symptoms		Fatigue, cyanosis and dysphagia	1 each
Pain in thorax (occasionally accompanied by paresthesia)	27	Physical findings	
Backache	3	Chest dullness	16
Lower extremity		Decreased breath sounds	11
Weakness, disturbance of gait or incoordination of lower extremities or all three	7	Asymmetry or scoliosis of thorax	14
Pain	2	Positive neurologic signs in lower extremities	7
Paresthesia	2	Portion of chest wall prominent	2
Upper extremity		Prominence of supraclavicular region	3
Weakness	1	Increased venous pattern of neck or upper chest	3
Pain	2	Horner's syndrome	3
Paresthesia	1	Skin nodules	6
Sphincteric weakness	1	Skin pigmentation (café au lait)	6
Cough	4	Paralysis of vocal cord	1
Sputum	1	Tender mass	1
Dyspnea on exertion	6		
Fever	2		
Loss of weight	2		

sult of mediastinal venous compression. Another complained of hoarseness of four years' duration and was found to have a schwannoma which involved the left recurrent laryngeal nerve.

All 8 of the thoracic tumors with intraspinal extension, the so-called dumbbell tumors, were symptomatic and first came to the attention of a physician because of symptoms referable to the spinal portion of the tumor. Thus, they presented with disturbances of gait, pain, weakness, paresthesia and incoordination of the lower extremities.

All of the 6 patients with malignant neurogenic tumors had symptoms. Besides symptoms due to pressure of the tumor on surrounding structures, a few of the patients had constitutional reactions to the tumor such as fever, loss of weight and fatigue.

Benign Schwannoma. A total of 86 cases of benign schwannoma was reviewed. Forty-five of the patients were females and 41 were males. The ages ranged from eleven to sixty-six years. Fifty-eight of the tumors were asymptomatic and were first noted from a routine roentgenogram of the thorax.

On gross examination the tumors were either round or oval and showed varying degrees of encapsulation. In most instances they were single, but in 6 cases the surgeon removed 2 tumors in close proximity to one another. In one instance the single tumor was bilobed. Six tumors were of the so-called dumbbell type. Some of the tumors had a smooth outer surface, but many were lobulated or presented nodules of varying size. The tumors ranged in weight from 5 to 2,135 gm. Approximately 40 per cent weighed more than 100 gm. (Fig. 1*a*).

The tumors in the fixed state were relatively hard and usually presented some resistance to cutting. The cut surface presented a varying appearance. Some of the tumors were solid but approximately a quarter of them were cystic. Rarely, the whole tumor was one large, single or multiloculated cyst with a granular, condylomatous interior and a thick wall. Nearly all the solid tumors showed evidence of degenera-

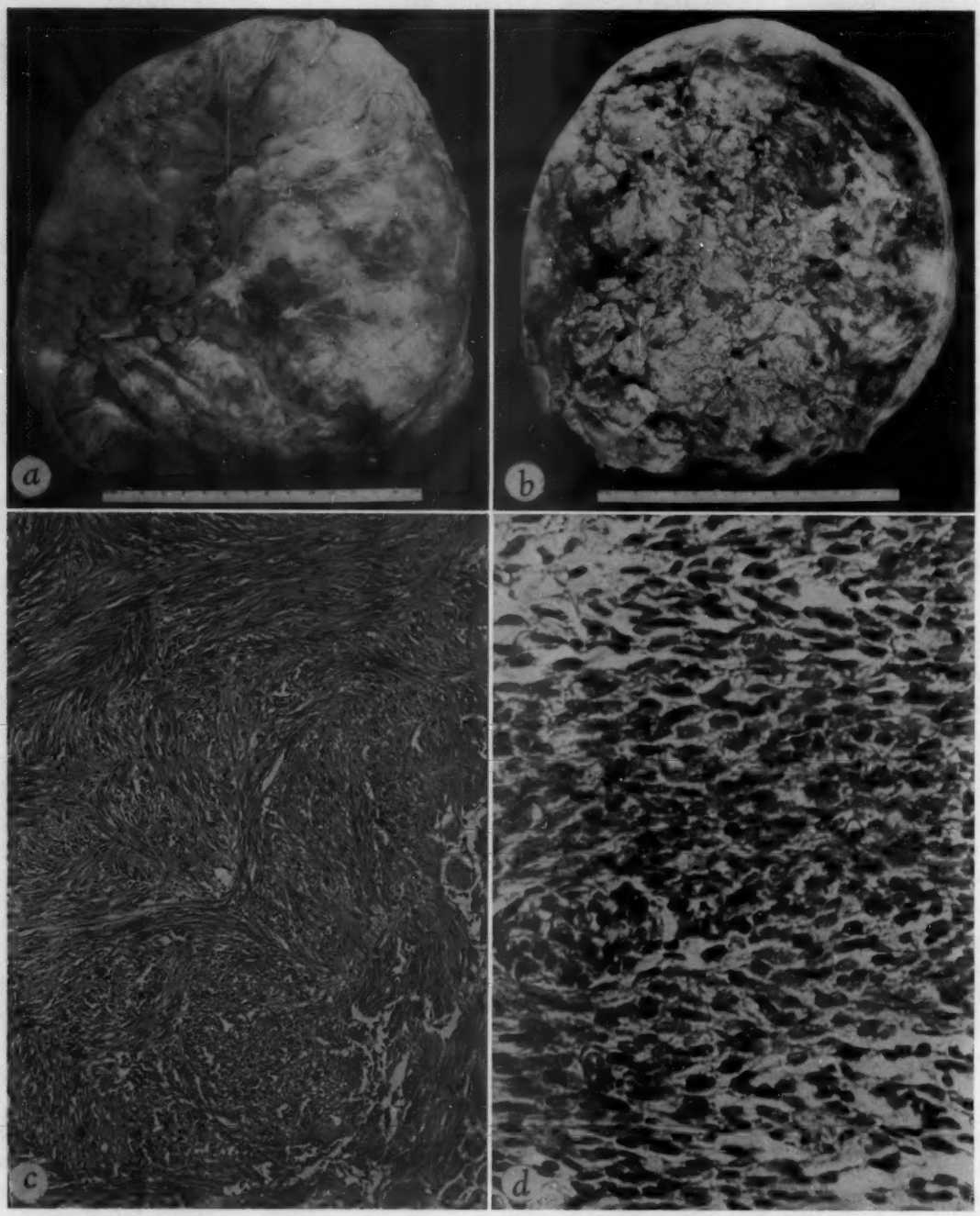

FIG. 1. Schwannoma. (*a*) Large encapsulated tumor. (*b*) Cut surface of the same tumor as *a*. The thickened wall, the marked hemorrhagic and fatty degeneration and the multiple cystic areas should be noted. (*c*) Typical microscopic pattern with interlacing bundles of Antoni A tissue separated by the loose Antoni B tissue (hematoxylin and eosin; ×100). (*d*) Microscopic appearance of a malignant schwannoma with very cellular tissue, palisading cells and numerous mitotic figures (hematoxylin and eosin; ×400).

tion of a fatty, hemorrhagic, hyaline or myxomatous nature (Fig. 1*b*).

Microscopically, the picture was characteristic, and usually two types of tissue were present, so-called Antoni A and B types (Fig. 1*c*). The more solid Antoni A tissue consisted of sheets and interlacing bundles of elongated spindle-shaped Schwann cells with palisading nuclei. Verocay bodies were seen occasionally. Antoni B tissue consisted of areas of loosely arranged Schwann cells, microcysts and reticulum fibers. One of the outstanding features was the presence of many thick-walled hyalinized vessels throughout most of the sections examined. Deposits of hemosiderin and cholesterol were seen rarely. Foam cells and focal collections of lymphocytes were common.

Several of the tumors deserve special mention. In 2 of them the microscopic pattern was a combination of schwannoma and neurofibroma, but the predominant feature was palisading Schwann cells. A third tumor was extremely cellular, but no mitotic figures could be found. Two others showed multinucleated Schwann cells, but again there was no evidence of mitosis. All of these tumors may be considered as atypical schwannomas.

Malignant Schwannoma. One case of malignant schwannoma was reviewed. A nineteen year old man presented with a lesion of the thorax known to have been present for nine years from roentgenographic examinations. He gave a one year history of pain in the thorax, dysphagia and loss of weight. He also showed the external stigmata of generalized neurofibromatosis with multiple cutaneous nodules and café au lait spots.

He underwent thoracotomy for removal of the tumor, but it was so invasive that it could not be removed. The biopsy tissue on microscopic examination was extremely cellular with closely packed palisading nuclei (Fig. 1*d*). Mitotic figures were easily demonstrated.

Ganglioneuroma. Thirty-nine cases of ganglioneuroma were reviewed. The ages of the patients ranged from three and one-half to fifty-nine years. Fifty per cent of the patients were less than twenty years old. Nineteen were females, and 20 were males. In 25 cases the ganglioneuromas were asymptomatic and the first evidence of the tumor was found on a routine roentgenogram of the thorax.

On gross examination most of the tumors were single with smooth surfaces and were either flattened ovoid or round (Fig. 2*a*). In one instance multiple tumors were fused into a single mass. In another case the tumor was bilobed, and in still another 2 discrete tumors were found side by side in the posterior mediastinal gutter. Two of the tumors were of the dumbbell variety.

For the most part the tumors presented fairly uniform characteristics in that they were usually soft, could be cut easily with a knife and possessed a glistening, homogeneous gray or sand-colored surface that felt slimy (Fig. 2*b*). However, a few were indistinguishable grossly from schwannomas.

Microscopically, the ganglioneuromas were variable, and their appearance depended on the degree of maturity (Fig. 2, *c* and *d*). On the one hand the section showed clusters of plump ganglion cells, the abundant cytoplasm of which stained an eosinophilic or violet color with hematoxylin and eosin. The majority of the cells were multipolar and usually contained one nucleus with a prominent nucleolus. Often the cell was surrounded by a clear zone and appeared to be lying within a lacuna. Everywhere throughout the section, a loose stroma of nerve fibers, usually of the nonmyelinated type, separated the groups of ganglion cells. Schwann cells were also present, and in rare instances definite proliferation and palisading of the nuclei were evident, making the pattern indistinguishable from that of schwannomas. In a few instances the stromal structures had been replaced by varying degrees of hyalinized connective tissue, and the ganglion cells were scarce, looked smudged, and contained hemosiderin in the cytoplasm. On

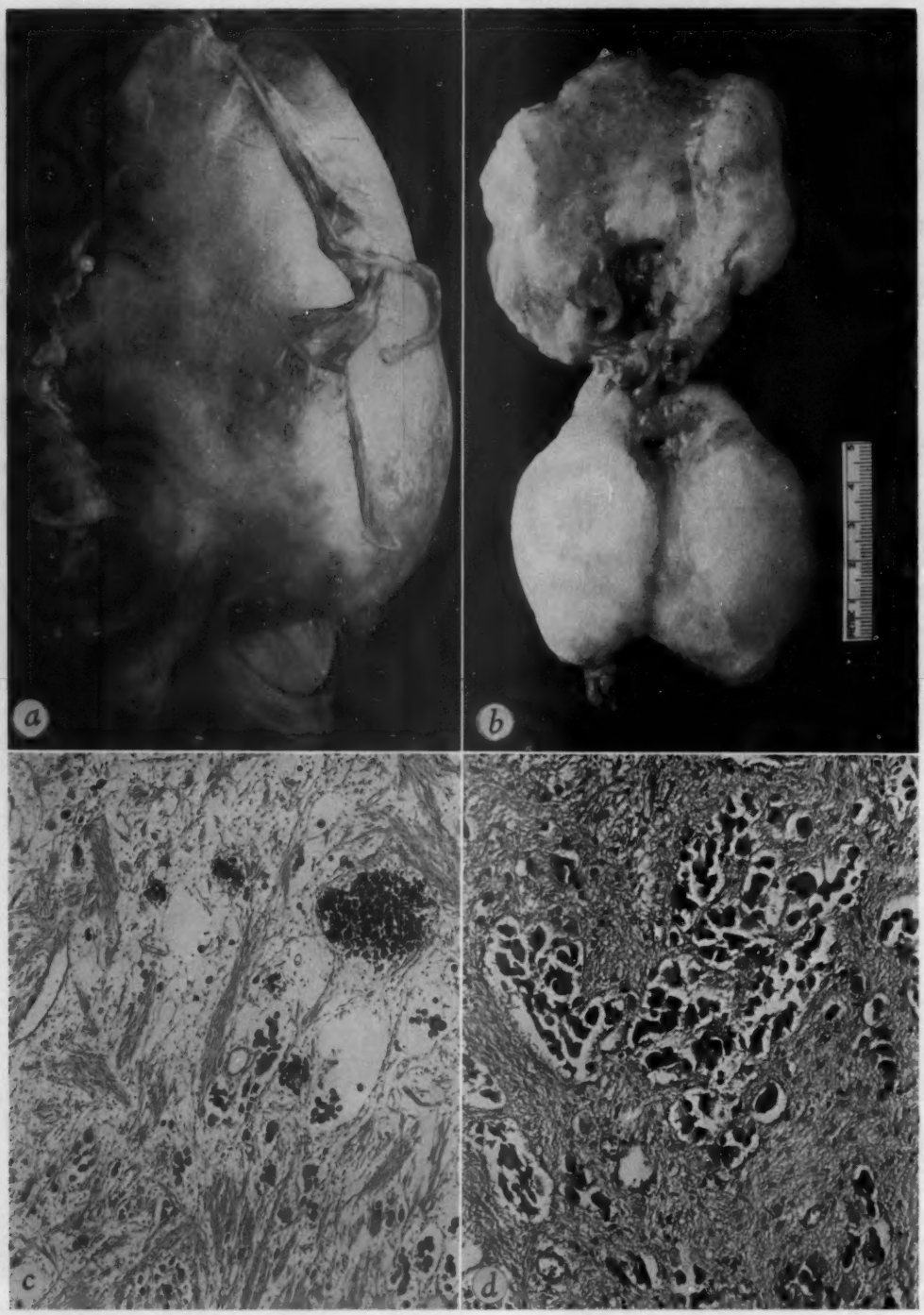

FIG. 2. Ganglioneuroma. (*a*) The tumor is ovoid and flattened, with a broad ragged base (not shown) where it was adherent to the mediastinum. (*b*) Cut surface of a ganglioneuroma showing the homogeneous glistening appearance and the lack of degenerative cystic changes. (*c*) Typical microscopic pattern of a ganglioneuroma showing the loose stroma of neurites, ganglion cells and foci of calcification (hematoxylin and eosin; ×50). (*d*) Immature ganglioneuroma with clusters of ganglion cells of varying shape and size. Many of the cells are multinucleated (hematoxylin and eosin; ×150).

the other hand were those tumors which were judged to be immature mainly on the basis of the appearance of the ganglion cells. The ganglion cells tended to be more numerous, to occur in tighter clusters or bands and to show a variation in size, shape and staining characteristics. The ganglion cells often contained more than one nucleus. No mitotic figures were seen. Ten of the tumors were considered immature ganglioneuromas. Two of these occurred in patients aged twenty-four and twenty-seven years, respectively. The remaining 8 were discovered in children whose ages ranged from three and one-half to nine years. One of the prominent microscopic features of the ganglioneuroma was the presence of small areas of calcification. Sixteen of the tumors showed calcium under the microscope. The occurrence or degree of calcification did not seem to be related to the maturity of the tumor. Approximately a third of the tumors showed focal collections of small, round, deeply-staining lymphocytes. No areas of neuroblastomatous change were noted in this series of tumors.

Neuroblastoma. Five neuroblastomas were studied. Three were in females and 2 in males. The ages of the patients ranged from one month to eleven years. Three of the 5 tumors were discovered before the patients were two years old. All of the patients were symptomatic. The duration of symptoms was relatively short, varying from one week to one year. Two of the patients complained of pain in the chest. Increasing dyspnea was noted twice. Fatigue and loss of weight were presenting complaints in one instance. Intermittent fever was exhibited by 2 patients.

Three tumors were intact enough to be examined grossly. One was indistinguishable from a cystic schwannoma except that it was of an odd reddish-brown color. Of the other 2, one was solid and encapsulated, and the other was irregularly shaped, nonencapsulated and presented a nodular exterior surface (Fig. 3*a*). Both tumors were soft and meaty. The weight of the tumors ranged from 15 to 110 gm.

Histologically, the most typical picture consisted of areas of small, closely packed, round cells with blue-staining nuclei (Fig. 3*b*). Rosette formation was noted occasionally, and feathery intercellular glial-like material and foci of calcification were present in all instances (Fig. 3*c*). Sections from 2 of the tumors showed them to be relatively acellular with necrosis, much hemorrhage and hemosiderin.

Neurofibroma. In the 9 cases of neurofibroma, 5 lesions were associated with generalized von Recklinghausen's disease, and each of the remaining 4 presented as a localized single tumor of the mediastinum. The patients with generalized neurofibromatosis were three, nine, nine, fifty and sixty-five years of age, respectively, and 4 of them were males. Only one had symptoms referable to the mediastinal tumor. He was a fifty year old man who had had thoracic pain for fourteen years, had known of an abnormal mediastinal density in the roentgenogram of the thorax for two years and had had weakness of the legs for six weeks. At operation extension of the tumor into the spinal canal was found.

Grossly, these tumors were characteristic and consisted of a plexiform mass of enlarged nerves (Fig. 4*a*).

Microscopically, all elements of a nerve trunk were present: sheath cells, neurites and connective tissue (Fig. 4*b*). The sections showed proliferation of Schwann cells unlike the pattern of growth seen in the schwannoma. The proliferating Schwann cells were accompanied by a diffuse, haphazard interlacing network of neurites. One noticeable feature was the "looseness" of the microscopic structure presumably due to the presence of interstitial edema. Replicas of the Wagner-Meissner tactile corpuscle were noted occasionally.

The 4 localized mediastinal neurofibromas not associated with clinical neurofibromatosis were microscopically indistinguishable from the plexiform neuro-

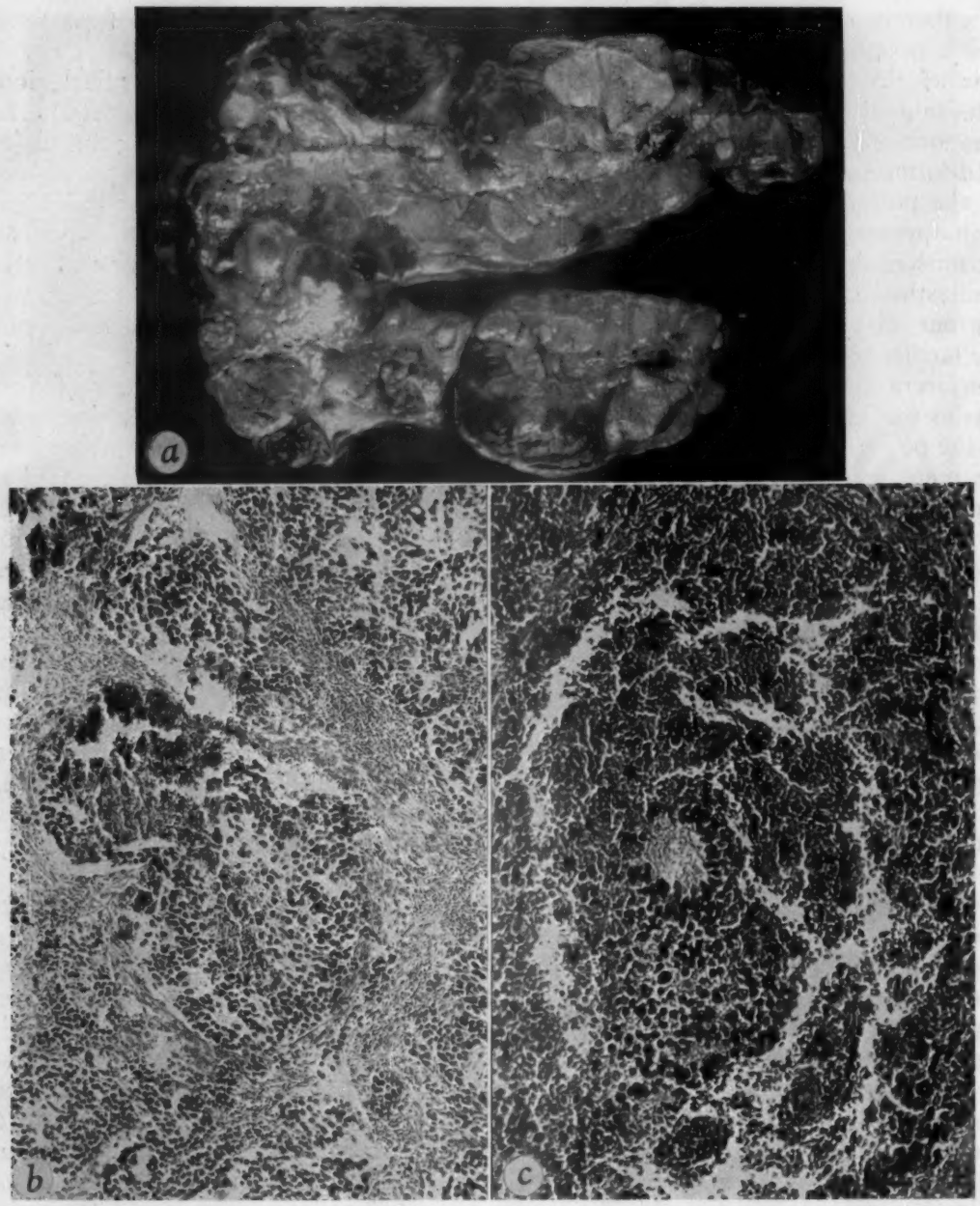

FIG. 3. Neuroblastoma. (*a*) Soft, nodular, meaty tumor with areas of hemorrhage. (*b*) Typical microscopic pattern showing small darkly stained, closely packed cells and foci of calcification (hematoxylin and eosin; ×100). (*c*) Rosette formation with central feathery glial-like material (hematoxylin and eosin; ×200).

fibromas just described. However, grossly there was no similarity. The localized tumors were round or oval, cut easily with the knife and had a myxomatous slimy surface without any evidence of degeneration.

ROENTGENOLOGIC FEATURES

The roentgenograms or photographs of the roentgenograms in 94 cases of mediastinal neurogenic tumors were examined. These included 62 cases of schwannoma, 23 of ganglioneuroma, 4 of neuroblastoma and 5 of neurofibroma. Three of the neurofibromas were plexiform in type and were associated with von Recklinghausen's disease.

Although it has been stated that neurogenic tumors do not have any character-

istic roentgenologic features that would enable a person to distinguish one type from another, this is not altogether true. The schwannoma and the ganglioneuroma, when they occurred in the superior and inferior mediastinum, were often indistinguishable. In the posteroanterior view of the thorax each appeared as a dense, rounded or oval circumscribed mass which blended with the mediastinal shadow medially but whose contour elsewhere was discernible from the mediastinal structures (Fig. 5, *a–d*). In the lateral view the tumors were defined just as well, and the mass always extended to the posterior limits of the intervertebral foramen except in the 3 cases in which the tumors arose from an unusual site such as the vagus or the recurrent laryngeal nerve. In some cases in which the tumor occurred at the superior limit of the mediastinum, the definition in the lateral view was poor. However, ganglioneuromas that occurred in the region of the mediastinum posterior to the hilar structures, the base of the heart and the root of the great vessels were usually characteristic and appeared different from all other neurogenic tumors. In the posteroanterior view the shadow cast by the ganglioneuroma in this region appeared as an elongated, flattened or triangular density with the broad base toward the mediastinum (Fig. 6, *a–d*). In many instances the density of the tumor faded off superiorly or inferiorly or both so that the limits of the mass could not be discerned. In the lateral view the tumors were characteristically poorly defined, and often there was only a suggestion of a density lying posteriorly without a definite border.

In one of the 3 cases of plexiform neurofibroma of the mediastinum associated with generalized neurofibromatosis, the tumor appeared as a flattened, somewhat lobulated mediastinal density (Fig. 7*a*). The other 2 tumors presented as a double contoured ovoid density in the superior mediastinum.

The 2 isolated mediastinal neurofibromas and the one malignant schwannoma (Fig. 7, *b* and *d*) presented roentgenologic appearances which were indistinguishable from that produced by the benign schwannoma.

Fig. 4. Plexiform neurofibroma. (*a*) Portions of a tumor removed from the mediastinum of a patient with generalized neurofibromatosis. (*b*) Microscopic pattern showing proliferation of Schwann cells in a loose interlacing network of neurites (hematoxylin and eosin; ×50).

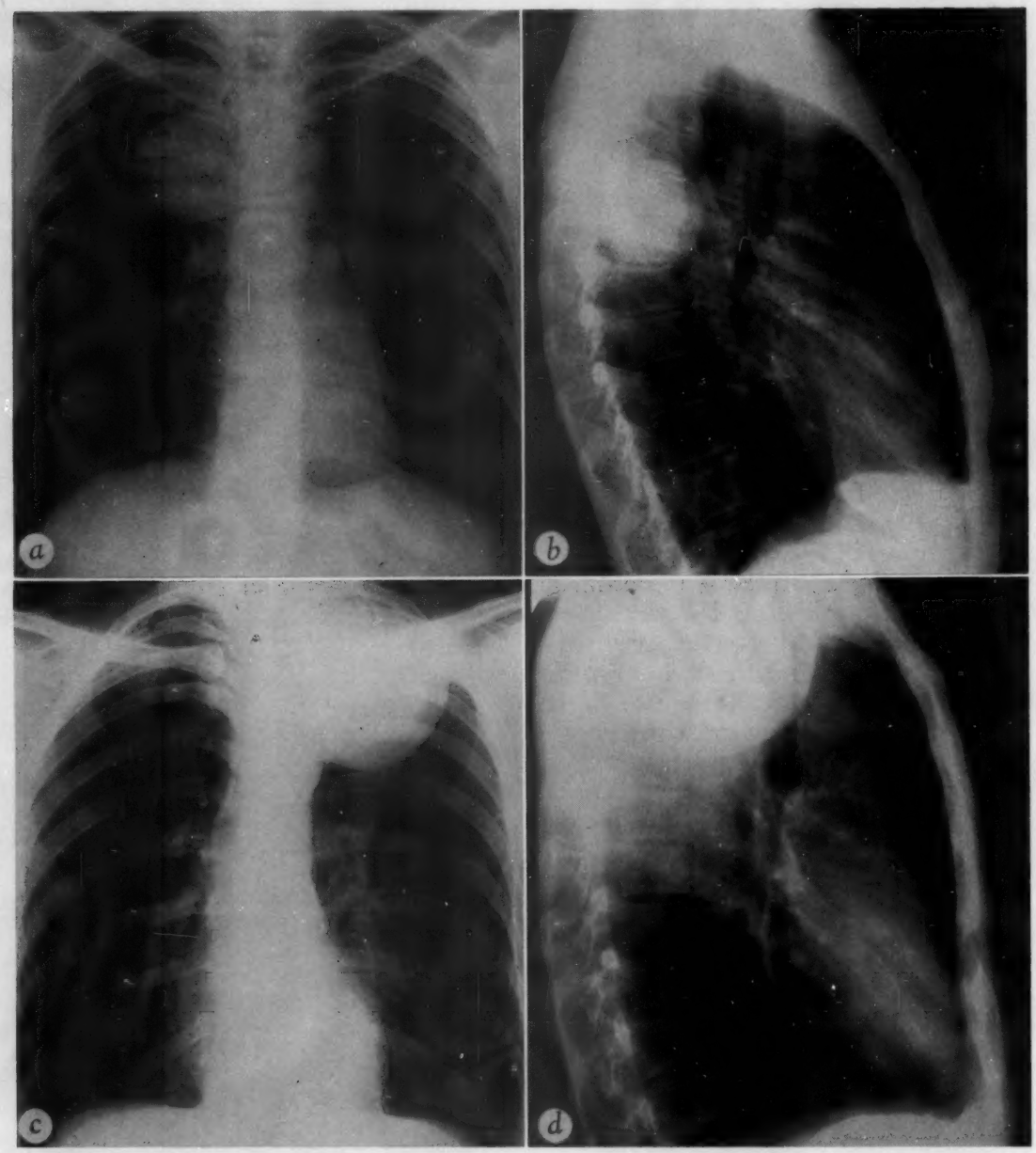

FIG. 5. (*a* and *b*) Typical, round, circumscribed, mediastinal schwannoma. (*a*) Posteroanterior projection. (*b*) Lateral projection. The tumor is seen as clearly as in *a*. It extends to the posterior limits of the intervertebral foramen. (*c* and *d*) Ganglioneuroma, situated in the superior mediastinum. (*c*) Posteroanterior projection. (*d*) Lateral projection.

The roentgenographic appearance of the neuroblastomas varied. In one case the neuroblastoma appeared as a paramediastinal mass associated with an expanding destructive lesion involving the rib contiguous to the mass. The remaining 3 tumors appeared as ovoid mediastinal masses which were indistinguishable from schwannomas. In one of these patients, a one month old child, the third interspace on the right was widened and the undersurface of the third rib was scalloped.

Calcium Deposits. As mentioned previously, microscopic deposits of calcium

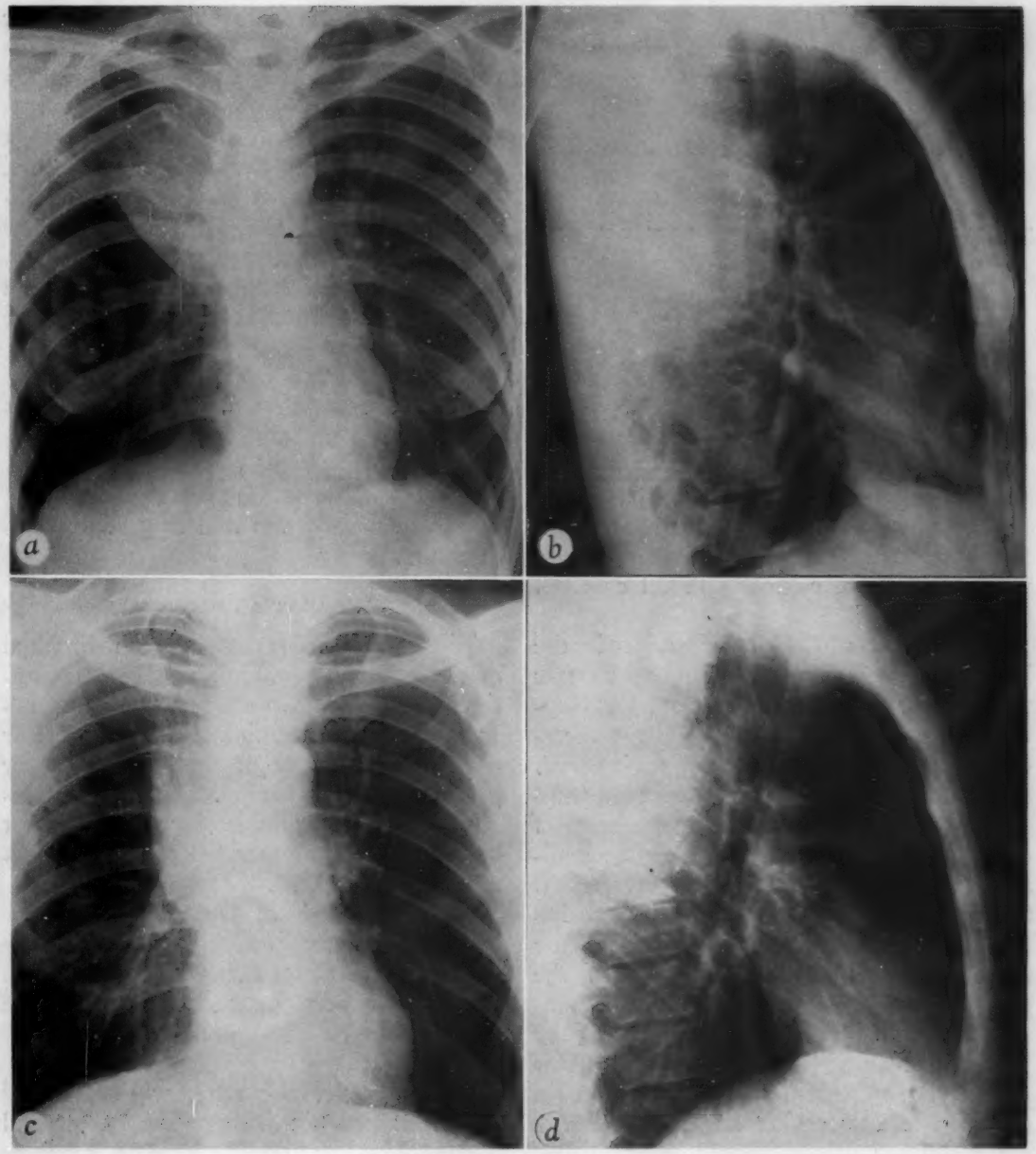

Fig. 6. Two examples (*a* and *b*; *c* and *d*) of a ganglioneuroma situated posterior to the root of the lung and great vessels. Note the elongated, flattened, broad-based appearance of the tumor and the manner in which it fades off superiorly. Characteristically, there is a lack of definition of the tumor in the lateral projection. Associated changes in the ribs and scoliosis also shown in *a*.

were common within the ganglioneuroma, but were discernible roentgenographically in only one such case. It appeared on the roentgenogram in that case as multiple small punctate densities (Fig. 7*c*).

Calcium was seen roentgenologically in 7 cases of schwannoma. In 6 it was deposited in a strandlike or curvilinear fashion in the walls of large or moderately large cystic tumors (Fig. 7*d*). In the other case the calcium appeared as multiple blotchy densities. Grossly, this tumor was markedly degenerated but not cystic.

Calcium was evident in the roentgeno-

TABLE IV

INCIDENCE OF SIGNIFICANT BONY CHANGES ASSOCIATED WITH NEUROGENIC TUMORS (NOTED BY EITHER ROENTGENOLOGIST OR SURGEON)

	No. of Cases
Ribs	
Erosion	39
Spreading	20
Destruction	1
Erosion of pedicle	13
Erosion of vertebral body	7
Enlargement of intervertebral foramen	7
Scoliosis	10

gram in one case of neuroblastoma. It was scattered diffusely throughout the tumor. As mentioned before, calcium, however, was noted microscopically in all cases of neuroblastoma.

Bony Changes (*Table IV*). Changes in the bones in association with neurogenic tumors were relatively common. Developmental anomalies of the ribs and the vertebral column were occasionally observed. Most of the changes seen in the roengenograms were brought about by pressure of the expanding tumor in the posterior mediastinal gutter or within the intervertebral foramen. The changes consisted of erosion and scalloping of the inferior portion of the vertebral end of the rib, erosion of the vertebral pedicles (Fig. 8*a*) and in some instances erosion of the lateral portion of the vertebral bodies (Fig. 8*b*). The anterior part of the vertebral body was eroded in one case. In an occasional case the pressure of the tumor was enough to spread and erode the vertebral ends of contiguous ribs and even cause a dislocation of the costovertebral junction (Fig. 8*b*). In several cases either a pedicle of tumor extended into the intervertebral foramen, or there was a frank dumbbell tumor. Under these circumstances there was evidence of erosion of the intervertebral foramen (Fig. 9, *a-c*). Actual destruction and invasion of bone occurred only in association with neuroblastoma.

One interesting feature of the neurogenic tumors was the association of scoliosis with the tumor (Fig. 8*c*). Scoliosis occurred in 7 cases of ganglioneuroma, practically a fifth of the ganglioneuromas. It was present in only 3 cases of schwannoma. In most instances the scoliosis was directed away from the tumor, but in a few cases it was directed toward the tumor. Generally speaking, the scoliosis associated with ganglioneuroma tended to be the more severe.

Displacement. Displacement of mediastinal structures, particularly the trachea or esophagus or both, was discovered occasionally during the course of roentgenologic investigation. It was noted in 15 cases of schwannoma and in 5 of ganglioneuroma.

DIFFERENTIAL DIAGNOSIS

The correct diagnosis of any mediastinal mass can often be made by noting the position of the tumor in the lateral roentgenogram and by determining the presence or absence of movement on swallowing or pulsation at fluoroscopy. The latter evaluation is often unreliable since one may not be able to tell whether the pulsation is direct or transmitted.

If the lateral roentgenogram of the thorax is arbitrarily divided longitudinally into three zones, an anterior, a middle and a posterior section, certain tumors are more inclined to be found in certain locations.

For example, the anterior mediastinal mass that does not move on swallowing and is situated at or near the base of the heart is likely to be a thymoma or teratoid tumor. Both tumors may contain calcium, but the finding of a tooth is pathognomonic for the latter. Thyroid adenomas, which also may appear in the anterior mediastinum, usually show evidence of extension down from the neck, and most important of all, usually displace the trachea and move upward on deglutition.

The middle mediastinum is often the site of enterogenous cysts which may be at-

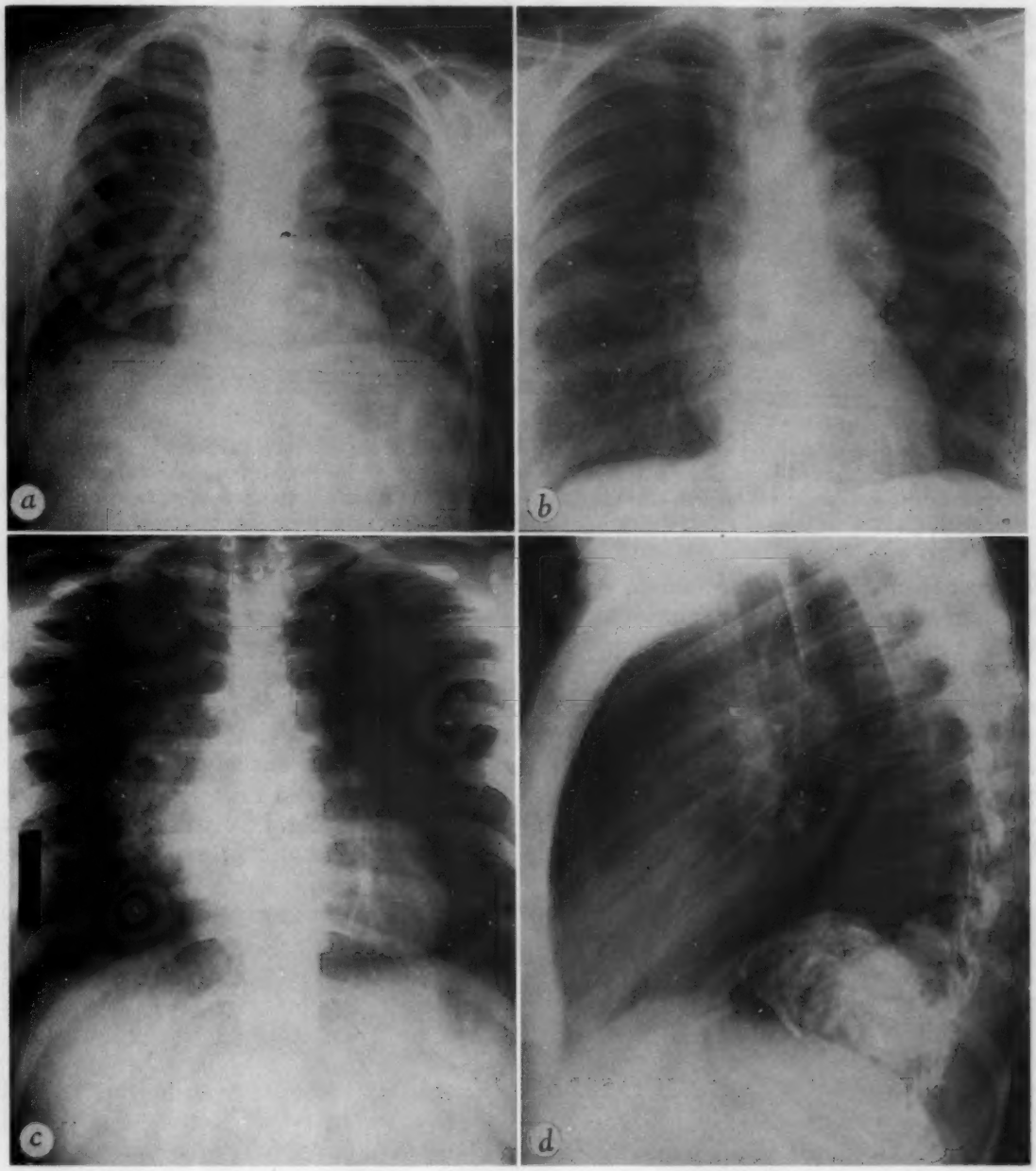

Fig. 7. (*a*) Generalized neurofibromatosis showing an elongated, lobulated mediastinal mass. (*b*) Two discrete tumors both of which were schwannomas. (*c*) Multiple punctate calcific densities in a ganglioneuroma. This was the only case of ganglioneuroma in which the calcium was discernible roentgenologically. (*d*) Multiple (2) lobulated schwannomas showing the strand and whorl-like calcification that may occur in such degenerative and cystic tumors.

tached to and deform the trachea and esophagus. Arterial aneurysms occur in this area, and if the diagnosis is not obvious at fluoroscopy, then angiocardiography may be needed. Mediastinal tumors of lymphatic structures such as lymphoma, sarcoid, and metastasis, when single, may be difficult to evaluate correctly, and the roentgenologist will require clinical data before he can suggest their identity.

Tumors which occur in the posterior mediastinum and extend to the posterior

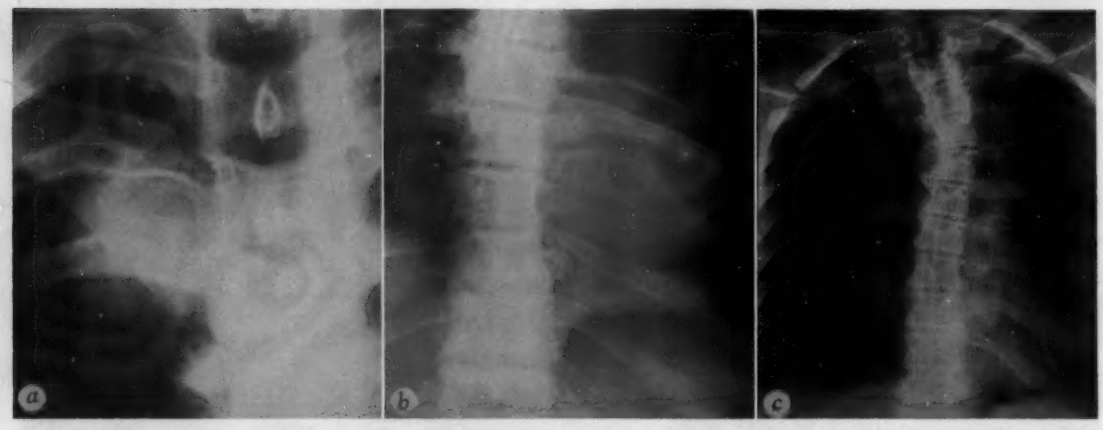

FIG. 8. Bony changes. (*a*) Typical rib changes that may be associated with a benign schwannoma. These consist of erosion, scalloping and sclerosis of the inferior aspect of the vertebral portion of the right third rib. (*b*) More advanced bony changes than in Figure *a*. The erosion involves several ribs and the lateral borders of several vertebral bodies. There is dislocation of the costovertebral junctions of two of the ribs. (*c*) Scoliosis is sometimes associated with neurogenic tumors, particularly ganglioneuromas.

limit of the intervertebral foramen, with or without bony deformity, are almost certain to be neurogenic tumors, and the ganglioneuroma situated in the posterior portion of the transverse middle third of the mediastinum presents a characteristic enough appearance to allow a correct diagnosis in most instances. In a rare instance the diagnosis will be in doubt, especially in cases of posterior mediastinal goiter, thoracic meningocele, and in cysts or tumors situated in the part of the parenchyma of the lung that lies in the posterior mediastinal gutter.

Those rare mediastinal tumors of neurogenic nature that arise from neural structures other than the intercostal nerves and paravertebral sympathetic chain such as the vagus nerve and that do not appear to overlap the vertebral column in the lateral roentgenogram show no distinguishing

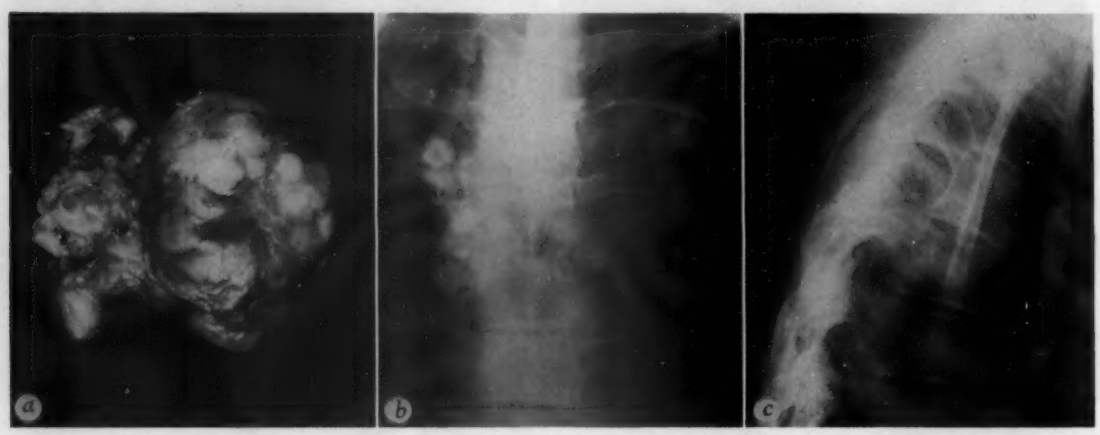

FIG. 9. Dumbbell schwannoma. (*a*) Gross specimen. (*b*) Posteroanterior Bucky roentgenogram of the thorax; same tumor as *a*. The erosion of the pedicle of the fifth thoracic vertebra on the left and the scalloping of the inferior border of the vertebral end of the fifth rib should be noted. (*c*) Lateral projection of the same tumor showing the erosion and enlargement of the corresponding intervertebral foramen. (*a*, *b* and *c* reprinted from Harrington, S. W. Intrathoracic extrapulmonary tumors: Diagnosis and surgical treatment. *Postgrad. Med.*, 1949, *6*, 6–21. [Author's and journal's permission received.])

characteristics to separate them from other cysts and tumors, which may occur in the posterior mediastinum.

SURGICAL CONSIDERATIONS

These tumors may be removed by an extrapleural or an intrapleural approach. In the early days of thoracic surgery the tumors were removed through a curving longitudinal posterior incision, the entire dissection and removal often being done without opening the pleural cavity. Today a routine type of posterolateral thoracotomy is usually employed. Surgical removal of the tumors in the series under consideration has resulted in 4 deaths during the immediate postoperative period, a mortality rate of 2.9 per cent. Three of these deaths occurred before or during 1930. One patient died thirteen hours after a combined procedure for the simultaneous removal of the spinal and thoracic portions of a dumbbell tumor. The cause of death was ascribed to postoperative shock. The 2 other patients died on the third and sixth postoperative days from pulmonary edema and bronchopneumonia, respectively. Only one postoperative death has occurred in 133 consecutive cases since 1930. This patient died of bronchopneumonia in 1937 on the eighth day after a combined procedure for removal of a dumbbell tumor.

The degree of difficulty encountered during removal of these tumors depended mainly on the type of tumor. The schwannoma was typically encapsulated and usually was enucleated easily. The ganglioneuroma, however, was characteristically an adherent tumor so that dissection was often extremely difficult and occasionally removal of all the tumor was technically impossible. The property of adherence was not solely a feature of the ganglioneuroma. In a few instances the schwannoma was adherent to contiguous structures; this was particularly true when the surrounding inflammatory reaction from a large cystic tumor obliterated the tissue planes. In several instances large schwannomas had caused collapse of the pulmonary parenchyma, and in 2 of these cases there were multiple fistulae between the compressed and adherent lung and the tumor.

FOLLOW-UP STUDIES

Schwannoma. Follow-up information indicated 4 deaths in the group of patients who had undergone removal of a schwannoma; 2 were from known unrelated causes and 2 from unknown causes. Three patients with schwannoma also had died during the immediate postoperative period. Thirteen of the patients could not be traced. This left a total of 66 for follow-up studies. All 66 were alive and well at the time of follow-up. Fifty-two of these patients had undergone the operation at least five years, and 31 at least ten years before the follow-up. Six were alive some twenty years after operation.

The tumor had recurred in 2 cases. Both of these tumors were so-called atypical schwannomas, not because of their recurrence but because of their atypical microscopic pattern.

One of these patients, a thirty-eight year old woman, had been operated on in 1944 for removal of a schwannoma which "was enucleated without difficulty." The spinal portion of the tumor had been removed a week or so previously. In 1950, about six years later, the patient returned with a recurrent intrathoracic tumor which was densely adherent to the chest wall. It was mobilized with difficulty and finally removed. She was last heard from in 1957 and was apparently well. Microscopically, the tumor was extremely cellular.

The other patient, a thirty-five year old woman, had undergone removal of a mediastinal tumor in 1946. The tumor which was adherent was removed with difficulty. Microscopically, the tumor was composed of elements of both schwannoma and neurofibroma. The patient returned in 1948 with a recurrent tumor. Thoracotomy revealed that the tumor was so adherent that removal was impossible. The patient returned home and subsequently received a course of deep roentgen-ray therapy. Her doctor wrote the clinic during 1958 and stated that she was "alive and quite well."

Neurofibroma and Malignant Schwannoma. One of the group of 5 patients with a mediastinal neurofibroma and generalized von Recklinghausen's disease could not be located. One had died postoperatively. Three were alive and well, two, five and eighteen years, respectively, after operation.

Two of the patients who had undergone removal of nonplexiform isolated neurofibromas could not be located. The remaining 2 were alive some six and sixteen years following the operations.

The patient who had a malignant schwannoma associated with generalized neurofibromatosis which was inoperable had returned home, but could not be traced.

Ganglioneuroma. Follow-up data were available from 38 of the 39 patients who had had ganglioneuromas. One patient died at the age of sixty-three years, four years after operation, presumably from some type of cardiac condition. All the others were alive. Twenty-eight of these patients have been traced for at least five years, 19 for at least ten years and 9 for fifteen years.

One patient had had multiple operations for recurrence.

This six year old girl came to the clinic with spastic paralysis and contracture deformities of the legs. Approximately four years prior to admission, she had had a series of three operations for removal of a tumor which presumably had occurred within the spinal canal and had extended into the right and left sides of the thorax. During her visit at the clinic a recurrent tumor was removed from the left side of the thorax. Microscopic examination of the specimen and a review of the slides of tissue removed elsewhere revealed a benign ganglioneuroma. The child has been followed now for about three years and to date has not had a recurrence.

In 5 instances all of the tumor could not be removed because it was so densely adherent. Only one of these patients received radiation therapy following operation. At least ten years had elapsed since operation on 4 of these patients and six years had elapsed since operation on the fifth. All were alive and well at the time of follow-up.

Neuroblastoma. Of the 5 patients who had had neuroblastomas, 2 had had recurrences with subsequent death, one approximately one year and the other five years following the initial operation. In both instances the surgeon was unable to remove all the tumor, and both patients had received postoperative radiation therapy.

The remaining 3 patients were alive and well. Two were given radiation treatment after operation. Both received approximately 2,200 r. The surgeon had been unable to remove all the tumor from one of these 2. These 2 patients have survived four and three years, respectively.

Of most interest is the third case. The tumor which was adherent to the surrounding tissues was "almost completely removed" from a child, eighteen months old. On original microscopic examination it was diagnosed as a malignant sarcoma of questionable origin. The child was given a grave prognosis and sent home without any radiation therapy. She was still alive and well at the time of follow-up some eleven years after operation.

COMMENT

The microscopic variability and the difficulty occasionally encountered in classifying certain neurogenic tumors have been well documented.[1,5] This is especially true of tumors that arise from the sympathetic nervous system. Several authors have described tumors which contain both ganglion cells and neuroblastomatous tissue. Such tumors are commonly referred to as ganglioneuroblastomas. Our own experience with neurogenic tumors of sympathetic origin suggests that the majority of the tumors can be classified as either ganglioneuroma or neuroblastoma and that the transitional type is uncommon. In the present series some tumors classified as ganglioneuroma did show varying degrees of immaturity based on the appearance of the ganglion cells, but we were unable to demonstrate any neuroblastomatous areas.

Recently, however, we examined a mediastinal tumor which was removed from an adolescent male. At operation the tumor was adherent to the mediastinum by a broad base, and appeared to be a ganglioneuroma. Microscopically, the sections showed a mixture of immature ganglion cells and neuroblastomatous tissue.

Thus, although the ganglioneuroblastoma is undoubtedly an uncommon tumor, it appears to have a place in the spectrum of tumors of sympathetic origin. Palisading Schwann cells may share in the neoplastic proliferation as was noted in one of the present cases of mature ganglioneuroma. In addition, several different types of neurogenic tumor may occur in the same individual as documented by Wahl and Craig.[6]

The ability of the tumors of the sympathetic nervous system to remain quiescent or regress is an interesting feature. Several such accounts appear in the literature.[2] In 5 cases in our series a portion of the ganglioneuroma was left behind without any apparent untoward effect. Four of these patients have been traced for at least ten years. One patient with a neuroblastoma has survived for eleven years since incomplete removal of the tumor without any postoperative radiation therapy.

Our study disclosed no evidence to support the view that these tumors may undergo a process of maturation.[4] What little data we have on patients who underwent multiple operations or who died of a recurrent tumor indicate that none of the tumors changed their identity over the period concerned.

The unique roentgenographic appearance of many of the ganglioneuromas is due to the characteristic elongated and flattened shape and the close adherence of the tumor to the mediastinum. Thus in the posteroanterior view the superior and inferior margins of the tumor may fade off, and only the lateral portion may show a definite outline. In the lateral projection none of the tumor may be in silhouette, and the definition of outline may be lost with the only evidence of a mass being an ill-defined area of increased density. In contradistinction the schwannoma is usually a rounded mass with a small base of attachment, and thus the tumor is in silhouette throughout most of its circumference and can be seen equally well as a well-circumscribed tumor in the posteroanterior and lateral views.

The surgical management of dumbbell tumors deserves special comment. In every one of our 8 cases, the presenting complaints were referable to the nervous system and were directly the result of spinal cord compression. We believe that the operation should be done in two stages, the spinal phase taking precedence. The thoracic portion of the tumor should be removed at a later date.

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LOCALIZED MEDIASTINAL LYMPH NODE HYPERPLASIA*

REPORT OF A CASE WITH ROENTGEN FINDINGS SIMULATING POSTERIOR MEDIASTINAL NEUROFIBROMA

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IN 1954 Castleman[6] described a patient with a large anterior mediastinal mass which on removal proved to be massive, localized, benign hyperplasia of lymph nodes, bearing a strong resemblance to thymoma grossly, microscopically and roentgenologically. By 1956 he and his colleagues[7] had collected 13 cases of this type, establishing a new disease entity. Most of the involved lymph nodes were in the hilar and tracheobronchial groups and appeared in the roentgenograms as sharply circumscribed hilar masses indistinguishable from the great variety of lesions found in this area. A few instances, in which anterior mediastinal lymph nodes were involved, presented as traditional multilobulated anterior mediastinal tumors. We have recently encountered a mass of the same histologic type involving a posterior parietal lymph node. Grossly and roentgenologically this simulated perfectly the classic neurogenous tumor of the posterior mediastinum. The roentgen features of this case are reported in order to call attention to the new entity of localized mediastinal lymph node hyperplasia, which should be considered in the differential diagnosis of tumors located not only in the anterior or middle compartments of the mediastinum but in the posterior compartment as well.

REPORT OF A CASE

A thirty-three year old white male was admitted for treatment of a mass discovered on a chest survey roentgenogram made one month prior to hospital admission on August 1, 1957. The mass was spherical in contour, homogeneously dense and measured 5.5 cm. in diameter. It was situated in the right paravertebral gutter and had produced slight erosion and pressure sclerosis of the inferior margin of the vertebral ends of the right fourth and fifth ribs (Fig. 1, 3 and 4). The roentgen findings were considered typical of neurofibroma or other neoplasm of neurogenous origin and no consideration was given to other lesions which might produce similar roentgen findings. There was also available a roentgenogram of this patient's thorax, made on November 8, 1948, approximately nine years earlier, as part of a mass chest survey. The latter roentgenogram showed the same mass, which at that time measured only

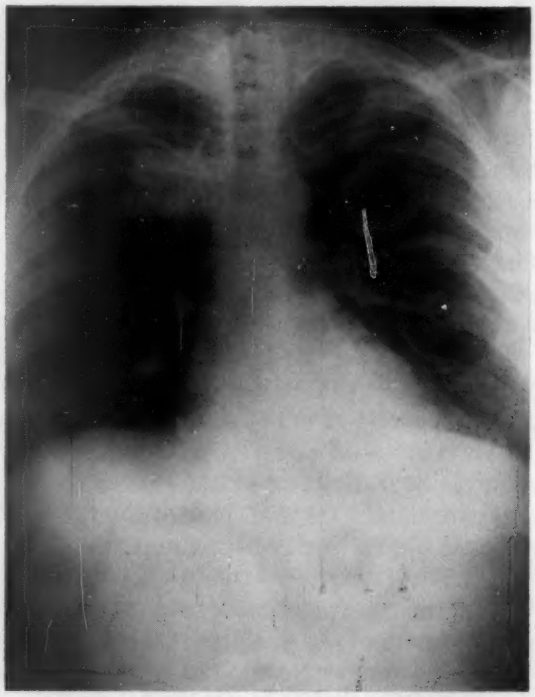

FIG. 1. Posteroanterior roentgenogram of the chest made on August 2, 1957, reveals a 5.5 cm. mass in the paravertebral gutter. A slight increase in density of the inferior margin of the vertebral end of the right fifth rib is noted in this view.

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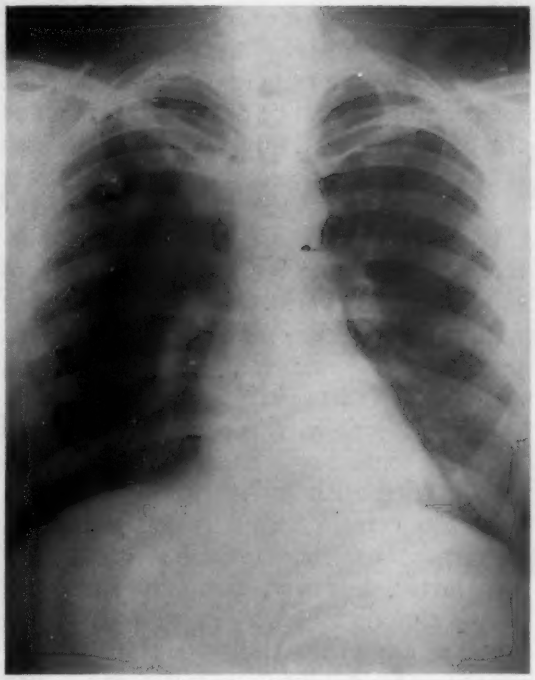

Fig. 2. The same mass is seen on a roentgenogram made approximately nine years earlier on November 8, 1948. At that time the tumor measured only 3.5 cm. in diameter.

3.5 cm. in diameter (Fig. 2). We could obtain no explanation as to why there had been no treatment of the mass at the time of its original discovery. Nevertheless, the roentgenograms furnished evidence of the extremely slow growth of the lesion, supporting the roentgen impression of a neurogenous tumor.

The patient was entirely asymptomatic and his past history, physical examination and laboratory studies were all unremarkable. On August 29, 1957, he underwent exploratory thoracotomy. A soft, solid, extrapleural mass, measuring 5.0×3.0×2.5 cm., was found adjacent to the third, fourth and fifth ribs posteriorly. It appeared to arise from within the third and fourth intervertebral foramina and was adherent to a short segment of the sympathetic chain. The tissue extending to the tumor from the intervertebral foramina and the adherent portion of the sympathetic chain was excised with the mass. The preoperative and postoperative surgical diagnoses were "probable neurofibroma." The patient made an uneventful recovery. He has remained symptom free and there is no evidence of recurrent tumor.

Examination of the gross specimen showed a soft, smooth, yellow-purple mass which measured 6.0×4.0×4.0 cm. The cut surface was yellow and showed hemorrhagic streaking. In one pole there was a small, discrete, yellow-white nodule, 1 cm. in diameter, standing out from the rest of the tumor.

Histologic sections revealed the mass to be an enlarged lymph node with prominent peripheral germinal centers. Compressed germinal centers were noted in the stroma and a large aggregate of typical lymphocytes was seen throughout. In several areas there were groups of blood vessels with moderately thickened walls which produced marked narrowing of the lumens. A few neutrophils and occasional eosinophils were seen. Hyalinized foci of varying size were noted in the parenchyma (Fig. 5 and 6).

The small yellow-white nodule was a sympathetic ganglion. The original pathologic diagnosis was "chronic inflammatory process of lymph node, etiology to be considered; Hodgkin's disease should be excluded."

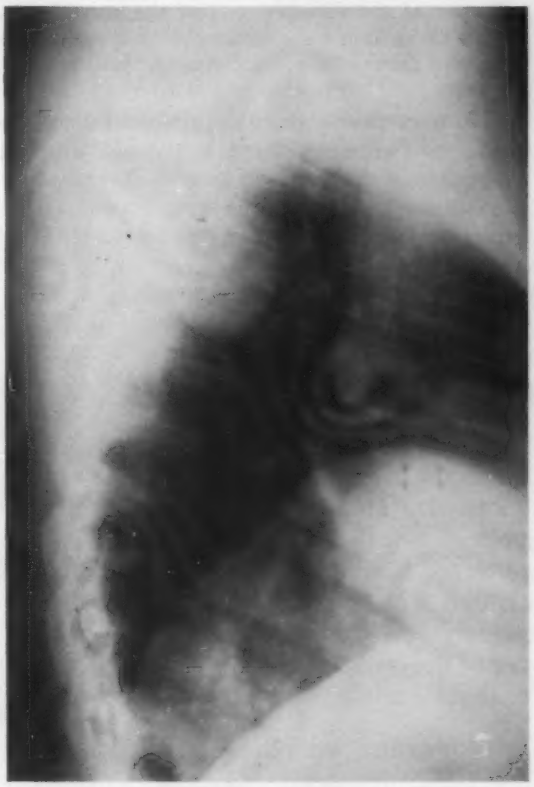

Fig. 3. The lateral roentgenogram of the chest reveals the mass in the extreme posterior portion of the thorax, lying in the angle between the dorsal spine and the ribs.

Fig. 4. Roentgenogram in the right oblique view of the upper chest shows the sclerotic, eroded inferior border of the vertebral end of the right fourth rib. The sclerotic change in the fifth rib is seen only in the frontal projection (Fig. 1).

The tissue was sent to the Armed Forces Institute of Pathology where it was identified as belonging in the group of tumors described by Castleman and his co-workers. It was coded under the heading of "massive chronic lymphadenitis."[20]

ETIOLOGY

The condition is regarded as a response to a chronic inflammatory process of long standing.[7] While Castleman *et al.*[7] did no bacteriologic investigations, they felt that a specific organism would not be found responsible. There is no evidence that the process is malignant since it has not been observed to recur locally or metastasize. Although 2 of their published cases had previously been reported as thymomas, they are of the opinion that the masses were not of thymic origin.

PATHOLOGIC CHARACTERISTICS

Grossly, the masses previously reported were found mainly at the lung root, near a large vessel or a lung fissure. Only 2 of the reported cases were found at the site of the normal anatomic location of the thymus. Our own case appears to be somewhat unusual in location although Castleman *et al.* mention one instance of a "4 cm. mass in the costo-vertebral angle." They have also seen a mass of the same size in the lower abdomen. The Armed Forces Institute of Pathology has recorded "isolated, tumor-like inflammatory lymph node masses of this type from various sites, superficial as well as deep, some reaching enormous proportions as, for example, one in the inguinal region which was the size of a grapefruit."[20]

Microscopically, there is hyperplasia of the lymphoid follicles and marked capillary proliferation with endothelial hyperplasia, which in certain arrangements present a pattern resembling Hassall's corpuscles. The latter, however, normally show the staining quality of keratin which was not found in any of the cases described. Eosinophilic and plasma cell infiltrations are also present.[7]

CLINICAL FEATURES

There appear to be no definite clinical symptoms manifested by the patients reported by Castleman and co-workers. Our patient also offered no complaints and his physical examination was negative. Almost all the cases were discovered during routine periodic or mass survey chest examinations. A few were detected when chest roentgenograms were made for complaints of frequent colds and coughs.[7] Symptomatic lesions due to bronchial compression have been observed.[20] The condition occurs in young and middle aged adults and in both sexes equally.

ROENTGEN FEATURES

As demonstrated in published roentgenograms, the hyperplastic lymphoid structures appear in the mediastinum as discrete, sharply circumscribed hilar masses resembling hilar lymph node enlargements, parenchymal lung masses or dilated vessels. A few appear as multilobulated anterior mediastinal masses resembling lymphomatous, thyroid, thymic or teratomatous

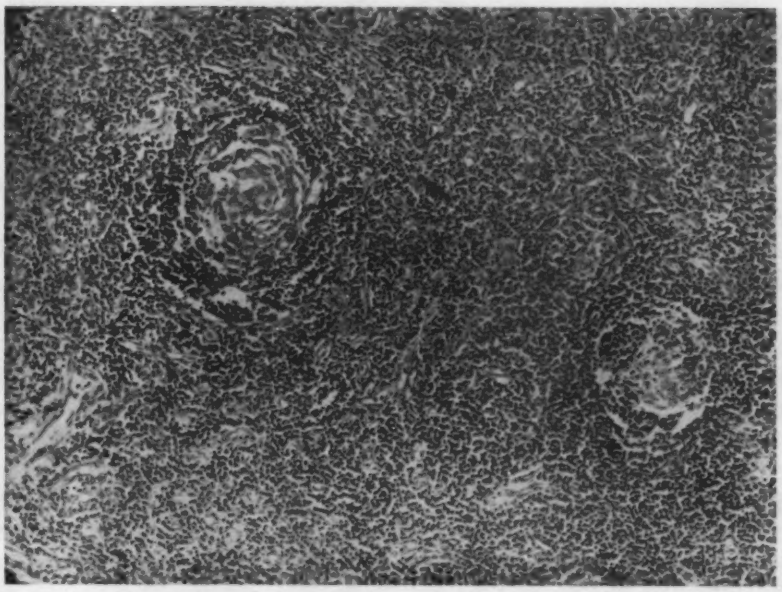

Fig. 5. Compression of germinal centers produces some distortion of the architecture of this lymph node. Hyperplasia of lymphocytes and hyalinized foci also tend to render the lymph node architecture less recognizable (×125).

tumors. At the operating table some have been mistaken for thymomas and others for bronchial adenomas. None have shown calcification roentgenographically or on histologic study.

In our own patient the mass had the typical roentgen appearance of a tumor of nerve tissue origin. This is understandable since it had apparently encircled the sympathetic chain and intercostal nerves, por-

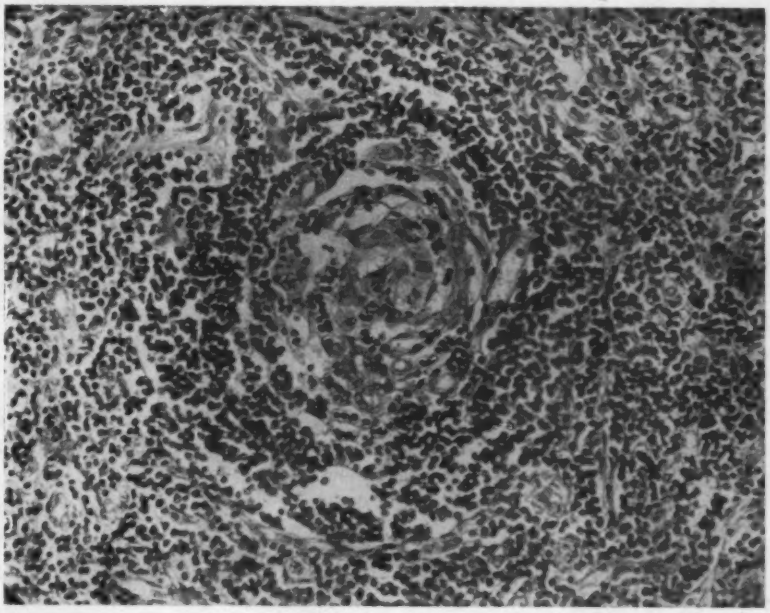

Fig. 6. Magnified view of follicle seen in upper left of Figure 5, showing hyalinized foci and vascularization (×325).

tions of which had to be resected with the tumor. The surgeon, too, was under the impression that he had removed a neurofibroma.

DISCUSSION

Our failure to appreciate the possibilities in the differential diagnosis of posterior mediastinal lesions prompted us to review the literature on this subject, which proved to be devoted largely to neurogenous tumors. These have been designated as virtually the sole occupant of this area. Neurogenous tumors have been described as: "More characteristic than any other group of mediastinal neoplasms . . . in fact there have been only one or two authenticated cases of nerve tumors occupying an anterior position";[21] "no other mediastinal tumor demonstrated such constancy of location";[25] "the appearance of neurogenic tumors is classic";[19] "posterior mediastinal tumors should be considered as arising from nerve tissue until proved otherwise . . . an extreme posterior mediastinal position and soft tissue density with sharp outline are classic";[8] " . . . the principal characteristic of the tumor is the position in the posterior mediastinum";[9] "almost universally in the posterior mediastinum";[4] "chiefly of one common variety";[3] "the roentgenologic evidence of a shadow in the posterior mediastinum in any age group regardless of absence of clinical symptoms should be considered as most likely due to a neurogenic tumor."[13] Textbooks of roentgen diagnosis repeat these statements. There is only one source which is at variance with these views. This is the work of Robbins,[22] who found that of 14 neurogenic tumors which he had studied only 9 were in the posterior mediastinum, while 5 were in the anterior mediastinum. It is also of some interest to note that as recently as 1941[15] one of the first papers dealing with the roentgen aspects of intrathoracic neurogenic tumors stated that, of the 7 cases reported, only 1 had been correctly diagnosed roentgenologically, although the roentgen findings in 6 were what is now regarded as "characteristic."

Even though it has become axiomatic that the histologic nature of a given mediastinal or thoracic mass cannot be predicted with absolute certainty from the roentgen findings, one should be particularly aware that "typical" roentgen findings associated with neurogenous tumors have been observed with lesions of other types. The literature contains a number of such cases. These lesions have presented as sharply circumscribed, spherical masses in the paravertebral gutter with nothing otherwise distinctive about their roentgen appearance. One should also be aware that 50 per cent of neurogenic tumors are not associated with skeletal erosion or sclerosis involving rib, vertebral body, intervertebral foramen or pedicle.[10] Such bone changes are merely highly suggestive of nerve tissue lesions but are not absolutely diagnostic. Our own case, although apparently rare, indicates that one may encounter a non-neurogenous tumor with associated erosive and sclerotic osseous changes. Rib and transverse process erosion were also present in the case of a unilateral tuberculous abscess of the thoracic spine which simulated a neurofibroma.[14]

The following are instances recorded in the literature where lesions of the paravertebral gutter, originally thought to be neurogenous, proved to be due to other causes.

1. Unilateral Tuberculous Abscess of the Thoracic Spine. In this report the authors[14] observed that the abscess "so closely resembled a spinal nerve neurofibroma roentgenologically that it seems worthy of record . . . that confusion of a tuberculous abscess with a spinal neurofibroma on roentgen study would seem unlikely, but that this is possible appears from the following case, most consistent with, and in fact almost diagnostic of, a spinal neurofibroma of the dumb-bell type." In this case the lesion had also produced erosion of the adjacent rib and the related transverse process.

2. Metastatic Carcinoma, Bronchogenic Carcinoma, Chondrosarcoma of a Rib and "Cyst." The authors[1] state that these four

lesions "have produced roentgenologic signs that have made them difficult to differentiate from a tumor of neurogenous origin."

3. Extrapleural Cyst. Two instances of mass lesions in the thorax which resembled neurofibromas roentgenologically are reported by Robbins.[23] One of these is described as a pleural cyst and the other as a bronchogenic cyst.

4. Endothelioma. This rather rare type of tumor is reported with the following comment: "endotheliomas originating in the posterior mediastinum in early cases may be difficult to differentiate from neurofibromas . . . in one case the pre-operative roentgenological diagnosis of neurofibroma was made."[11]

5. Lipoma. This lesion was described as "simulating a ganglioneuroma (or) neurofibroma, but other tumors could not be ruled out."[26]

6. Carcinoma of the Thyroid. In this instance "the appearance and location of the mass suggested a tumor of a nerve tissue origin."[2]

7. Anaplastic Bronchogenic Carcinoma. The author[17] comments that this mass was "thought to be a mediastinal neurofibroma."

8. Intrathoracic Meningocele. Five cases are cited by various authors[5,12,16,24,27] to illustrate the frequency of an erroneous preoperative diagnosis of neurofibroma in the presence of an intrathoracic meningocele. Teng and Eastman[27] recently reviewed all of the reports on intrathoracic meningocele since 1933 and found that in 11 of 24 cases the erroneous diagnosis of neurofibroma was made. The differential diagnosis of these 2 conditions is important and may be made by myelographic study or transthoracic aspiration of the cyst. A correct preoperative diagnosis may eliminate entirely the need for surgery since asymptomatic meningoceles need not be removed.[27]

9. Myxochondroma of the Chest Wall. Here the tumor "could not be distinguished from a neurofibroma radiographically."[18]

Inasmuch as our review of the literature did not include all of the material, particularly that available in foreign language journals, it is quite likely that additional instances exist in which an incorrect roentgen diagnosis of neurogenous tumor has been made, even though the number of such occurrences might prove to be small. It is highly probable that any one of the commonly occurring solitary pulmonary nodules such as the various granulomas, the hamartoma or bronchial adenoma might be discovered in the posterior paravertebral gutter, although we have not encountered any examples of this type.

SUMMARY

The entity of localized, mediastinal lymph node hyperplasia, thought to be the result of a benign inflammatory process of undetermined etiology (Castleman), is reviewed. A case is reported of a patient in whom the involved tissue presented as a posterior mediastinal mass simulating a neurogenic tumor. The roentgen features of this condition, and of posterior mediastinal masses in general, are discussed.

Instances are cited to show that the typical posterior mediastinal neurogenic tumor may be simulated, as in this case, by a variety of lesions. Thus the roentgen findings of a sharply circumscribed mass in the paravertebral gutter, with pressure erosion and sclerosis of an adjacent rib or vertebral body, are merely suggestive of a neoplasm of nerve tissue, but the radiologist cannot offer this diagnosis with assurance.

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UTILIZATION OF ROENTGENOLOGY IN THE STUDY OF SPEECH MECHANISMS*

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NEW roentgenographic equipment and techniques permit demonstration of structures and analysis of movements which previously were inaccessible to study. Study of the mechanisms and pathology of speech is increasingly utilizing roentgenographic methods. Recent publications in the field of speech pathology emphasize the usefulness of the roentgenographic examination and the important contribution it may make.[4,7,11,13] The radiologists, therefore, may expect increasing requests for their services and facilities in speech investigation. This paper describes equipment, techniques, and methods of analysis appropriate to this type of study.

Few radiologists have had clinical or academic contact with the speech pathologist and will have little understanding of his training or work. The profession of speech pathology includes both speech therapists and speech pathologists, with the latter designation being associated with a doctoral degree. Both groups are represented by the American Speech and Hearing Association. Their training includes specialized courses in speech and hearing, with additional work in clinical and experimental psychology, anatomy, and physiology. Speech pathologists are affiliated with universities, larger public school systems, rehabilitation centers, and speech and hearing clinics. Their activities include teaching, research, participation in clinical activities, and supervising the work of speech therapists. Often, speech rehabilitation centers will utilize the services of medical specialists, such as pediatricians, otolaryngologists, psychiatrists and others, and it is from such specialists that the radiologist may expect referral of patients for study. The usefulness of the radiologist in clinical or research studies of speech is well established. His training and experience qualify him to perform the necessary roentgenographic examinations and to assist the speech pathologist or the medical specialists in their interpretation and evaluation. Radiation safety and protection are particularly his concern.

ROENTGENOGRAPHIC EQUIPMENT

The standard diagnostic roentgenographic unit will provide still roentgenograms which are important in preliminary identification and demonstration of gross structural disturbances. As described below, such examinations will be of "poses" or static positions, which can be maintained only briefly. Thus, short exposure times are necessary. As a result, the roentgen-ray generator should have an output of 300 or 500 ma., a kilovoltage limit of at least 120 kv., and a timer extending to 1/60 or 1/120 of a second. To permit examination in both supine and erect positions, the roentgenographic table should have a 90 degree range of motion, horizontal to vertical. Since reduction of patient exposure requires the use of high speed film and intensifying screens, the roentgenographic unit should be equipped with at least a 12:1 grid to retain detail. The inherent high contrast of this film-screen combination permits the use of higher kilovoltage techniques.

Development of the image amplifier and cineroentgenographic equipment represents the most significant roentgenographic advance in demonstrating the rapid motions characteristic of speech. The excessive radiation exposure associated with earlier methods of motion study is no longer en-

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countered with modern equipment, and examination exposes the subject to no more radiation than during routine diagnostic fluoroscopy.

The combination of image amplifier, closed circuit television pickup, and sound recording is one that is particularly suited to the study of speech, although certain technical problems await solution before such equipment can be recommended. The television circuits permit alteration and adjustment of image contrast and density external to the image amplifier. Precise synchronization of sound and film strip is also possible in such apparatus by the use of 35 mm. optical sound film cameras. Field size of later models of cinefluorographic equipment is sufficient to include the entire pharyngeal and laryngeal areas.

In the installation of the image amplifier, sufficient clearance between the fluoroscopic tower and the table top must be provided to permit full lateral examinations of adult patients. Some units will not accommodate the width of the adult shoulders without alteration.

Slow motion photography of the amplified fluoroscopic image necessitates the use of frame speeds of 30 or more per second; most modern units provide such a range of speeds. Because of the delicacy of the negative film emulsion, duplication of the examination on durable positive film is recommended when frequent projection is anticipated. Film splices will also be protected by duplication.

Rapid film or cassette changers provide roentgenographic information and structural details not demonstrated by the image amplifier. They may partially compensate for a lack of cine-equipment. Biplane units have the advantage of providing simultaneous exposures in two planes, which may be desirable in some examinations. Figure 1 illustrates the usefulness of such rapid film sequences in demonstrating structural abnormalities and their effect on pharyngeal function; however, the number of individual frames obtained during such an examination is necessarily limited, and the time interval between exposures is significant in some rapid speech motions.

Laminagraphic sections, particularly as obtained with multilayer cassettes, are of special value in visualizing the laryngeal area. The true and false vocal cords can be identified only with difficulty in standard anteroposterior roentgenograms but are well shown by laminagraphy (Fig. 2). The prolonged exposure limits study to static poses or sustained phonations.

Accurate mensuration of pharyngeal structures and landmarks is usually required. Though the orthodontic cephalostats are useful for such purposes, the standard roentgenographic head unit or diagnostic unit will suffice if care is taken in positioning the subject.

ROENTGENOGRAPHIC TECHNIQUE

Standard roentgenographic techniques may not be suitable for studies of the speech mechanism. Both the density and contrast of the roentgenogram may require alteration to visualize particular areas or landmarks. For example, soft tissue contrast in the pharyngeal area of infants and children is increased by using a lower kilovoltage technique in cinefluorography and still roentgenography. Variation from standard positions may also facilitate demonstration of a particular structure or action.

To delineate soft tissue outlines, contrast material may be necessary. Iodized oil instilled through the nostrils is useful to demarcate the palate and upper pharynx. The tongue may be outlined by applying barium powder or paste, preferably after blotting or drying the tongue.

RADIATION PROTECTION

Clinical or research investigations which include roentgen-ray examination require adequate protection of the patients and personnel. The radiologist bears the responsibility of keeping radiation exposure at a minimum, while insuring that the examinations provide the desired information.

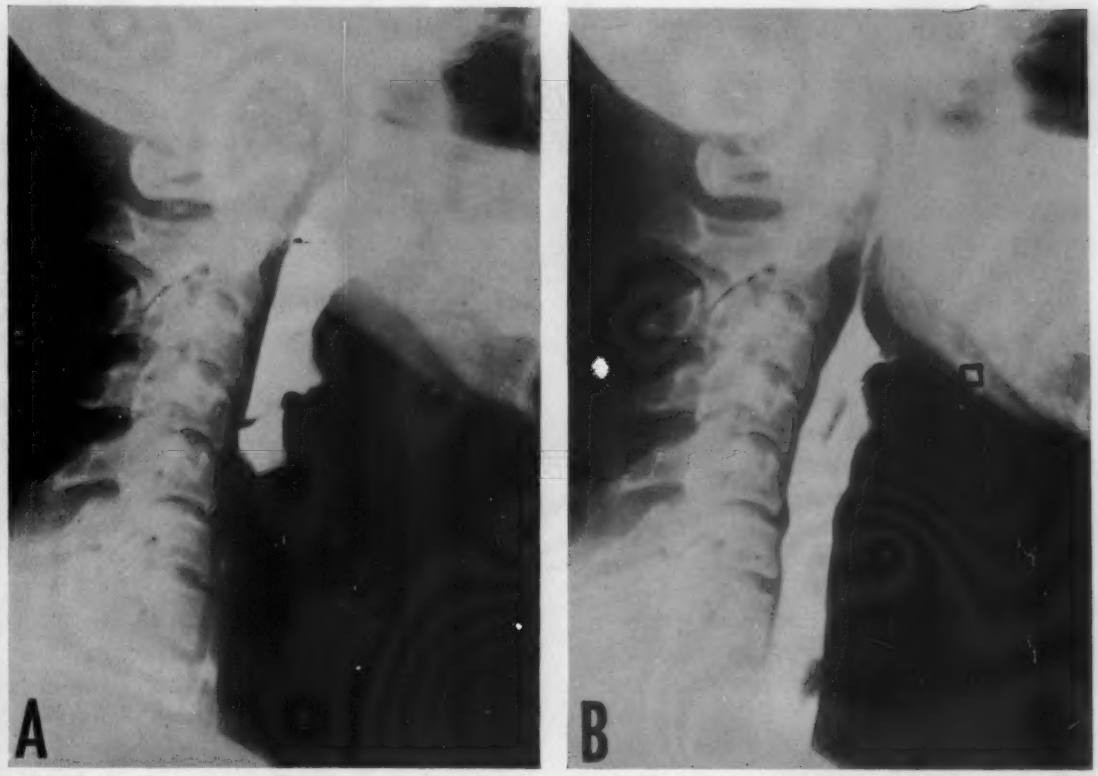

Fig. 1. Rapid sequence studies. Two selected roentgenograms are shown of a sequence obtained during the study of normal swallow. (*A*) The bolus of barium is in early swallow position. (*B*) Progress through the hypopharynx and cervical esophagus. The small square in *B* marks the position of the hyoid bone. Note the good structural detail and ready identification of landmarks.

Protective devices should be a part of the roentgenographic equipment. The fluoroscopic tube used with the image amplifier can be filtered with 3 to 4 mm. aluminum without significant loss of detail or contrast. In typical equipment, dosage rate with such filtration during cinefluorography of the pharynx and larynx is 5 r per minute in air at the table top at 30 frames per second, a rate which compares favorably with

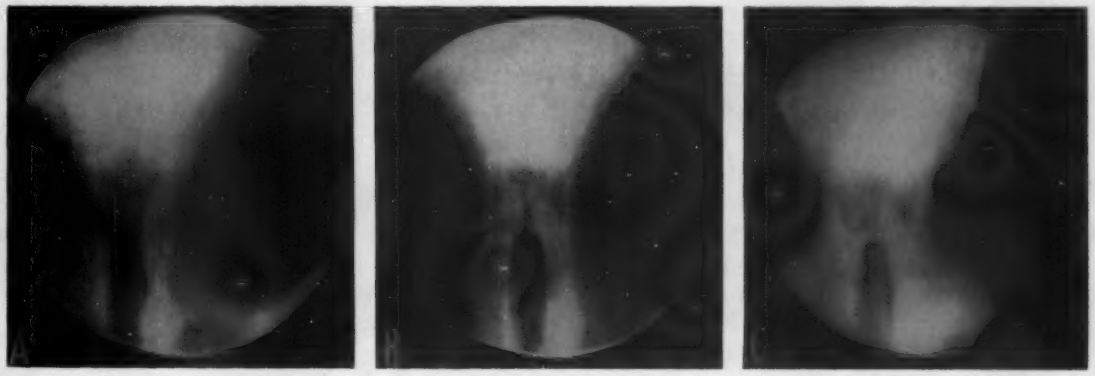

Fig. 2. (*A*) Laminagraphic section obtained in rest position—quiet respiration. (*B*) Phonation of normal subject. Note excellent visualization of vocal folds. Compare with abnormal phonation (*C*) with its deficient closure of false folds and gross abnormalities of the adjacent air cavity.

that delivered during a standard fluoroscopic examination. The beam should be sharply limited by the lead shutters to the field of the image amplifier.

The overhead tubes used for the still roentgenograms, laminagrams, and rapid sequence studies should also be filtered with 3 mm. aluminum. Multileaf collimating devices for coning are likewise effective in reducing total patient exposure and increasing definition and contrast. Exposure may be further reduced in laminagraphic studies by use of the multilayer cassette. The image amplifier itself represents a significant reduction in exposure as compared with earlier methods of motion study.

External lead shields, barriers, and aprons should be available to both subject and operating personnel. The gonadal areas require specific attention and protection during roentgenography. Duplicate records of exposure should be maintained in the radiology department and in the records of the speech pathologist. Periodic review of the examination procedure will prevent duplication or overlap of studies.

FILM ANALYSIS

Film analysis and accurate measurement of the speech structures will usually be the responsibility of the speech pathologist. The radiologist, however, must be prepared to give assistance and advice concerning any phase of the interpretation. In particular, identification of anatomic structures, evaluation and measurement of image distortion, and alteration of position or technique to demonstrate more clearly a movement or structure will be his contribution in a cooperative study. The extent of this contribution will be increased as the radiologist becomes more familiar with speech pathology. Specific methods of measurement and analysis have been described[5,13] and can also be applied to other physiologic functions.

In general, however, the motion picture strip is the most useful roentgenographic study, although analysis is a time-consuming process of frame by frame comparison. In such interpretation, tracings of frame sequences permit demonstration of isolated anatomic parts in motion and the relation of these parts to fixed landmarks. Composite overlays of complete motions or maneuvers provide comparison of the normal and abnormal patterns of speech and characterize the specific abnormality involved. In the absence of normal criteria of function, matched normal subjects may be examined, permitting comparison of one strip with another. Figure 3 illustrates the use of both individual and composite overlays. As is shown in Figure 3, the still roentgenogram is used to orient the film strip and aid in identification of the structures. Accurate measurement of structural movements and deviations from normal is also possible with the larger film and is preferable to subjective evaluation. For such measurements, calipers, rulers, protractors, and some special map-measuring instruments are used. To insure accuracy and duplicability, carefully standardized body positions should be assumed during the examinations. Prominent landmarks, such as the sella turcica, should be employed as points of reference in measuring structural displacement from the rest position, which is one of quiet respiration.

Figure 4 illustrates a tracing cabinet which has been designed for projecting and tracing cine film strips. Special projection equipment for this purpose is available from manufacturers supplying the cinefluorographic equipment. It should provide for prolonged projection of single frames without damaging the film, slow motion projection, frame counting, and remote control.

SPEECH PROBLEMS

Increasing use of roentgenography in clinical and research study may be expected as its value in diagnosis and evaluation of therapy becomes more generally appreciated. The radiation hazards are not excessive if suitable protective measures are employed. Nevertheless, examinations should be performed only to provide

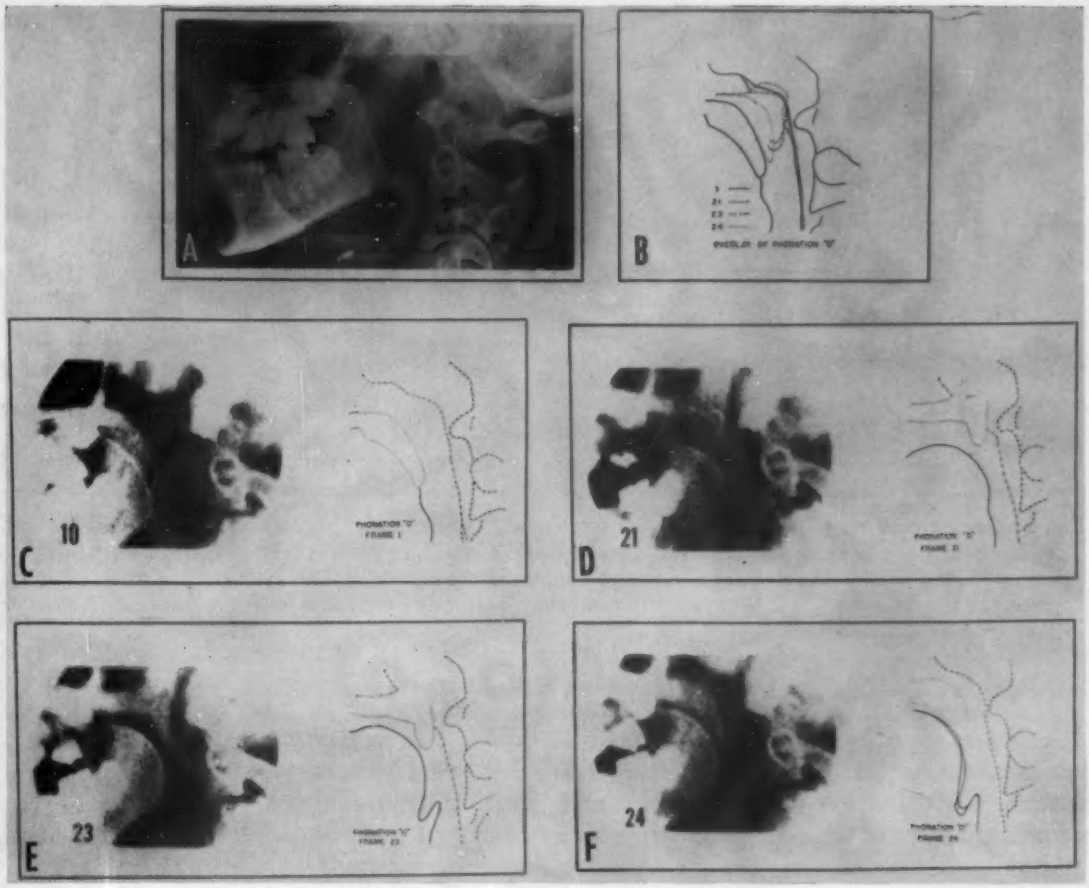

Fig. 3. Use of overlays. (*A*) Reference still roentgenogram in identical lateral position with the field of view of the cine strip indicated in the darker central area. (*B*) Composite overlay of phonation "O." (*C*, *D*, *E* and *F*) Individual frame overlays and reproduction of cine frames from which the composite overlay was constructed. Note the use of the bony landmarks as reference points.

information that cannot be obtained by other methods. The current and potential applications of roentgenology to this field are briefly summarized:

1. *Normal Speech.* Limits of normality have not as yet been defined for many of the structures and functions of the speech mechanism. Speech performance, particularly in infancy, is incompletely understood.

2. *Speech Disturbances Secondary to Unusual Variation in Normal Structures.* Certain combinations of otherwise normal structures may result in abnormal speech; for example, a large pharynx combined with a small soft palate, though each is ostensibly normal, may result in a hypernasal voice.[10] Specific identification of such extremes of normality may be accomplished only by roentgenographic observations.

3. *Structural Anomalies.* Anomalous development of the speech mechanism may result in disturbances of voice and articulation which may be readily identified and characterized by roentgenographic methods.[6,7,12,14]

4. *Compensations for Organic Disability.* Individuals vary in their ability to compensate for structural or functional defects. Study of such patients may help identify the successful methods of adaptation, which may then be applied to development of therapeutic techniques of value to other patients.

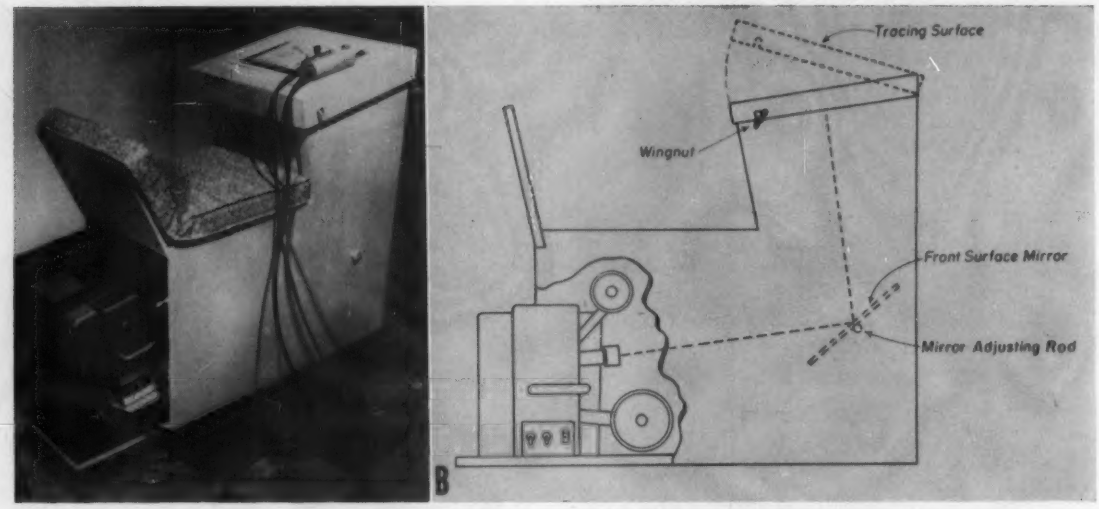

FIG. 4. Tracing cabinet. (*A*) Photograph of the cabinet. (*B*) Schematic diagram of its inner construction. Incorporation of the chair and backrest into the cabinet structure permits long periods of work without fatigue. The device to the right of the tracing cabinet is the remote control. The tracing surface is clear glass, the tracing paper itself being used to form the image. (By permission from the *Journal of Biologic Photographic Association.*)

5. *Identification of Functional Speech Disabilities.* Speech may be abnormal because of faulty learning or emotional disturbance. Evaluation of such effects and demonstration of normal basic structure and function may require roentgenographic examination. Comparison of voluntary and involuntary activities will often help in identifying purely functional disturbances.

6. *Primary Muscular Disorders.* Specific alterations in the speech mechanism associated with muscular dystrophy and related disorders have as yet been incompletely studied. Roentgenographic study is particularly suited to show changes in muscle mass or strength.

7. *Neurologic Disorders.* The patterns of dysfunction resulting from primary neurologic diseases are receiving increased emphasis; for example, poliomyelitis has been studied in detail, with considerable information being obtained by roentgenographic examination.[1–3] Other neurologic abnormalities of the speech mechanism lend themselves equally well to roentgenographic investigation.

8. *Effects of Tissue Destruction.* Identification and evaluation of specific effects of trauma, malignancy or destructive disease may be of primary importance in corrective speech therapy. Defective speech resulting from surgical procedures of the head and neck regions can also be studied by these methods.

9. *Evaluation of Medical and Surgical Therapy.* Medical treatment, dental prostheses, and surgical procedures designed for correction of structural abnormalities of the speech mechanism may be most objectively evaluated by roentgenographic methods. These supplement less accurate subjective conclusions of patients and physicians. The design and placement of prosthetic devices, such as obturators, may also be greatly facilitated.[8,11]

10. *Evaluation of Speech Therapy.* Serial studies of the individual patient can aid the speech pathologist in evaluation of specific therapy. Although such examinations will be limited because of radiation exposure, evaluation of accepted methods and suggested modifications can be accomplished.

SUMMARY

The current rapid advance in roentgenologic knowledge and equipment will lead

to increasing use of this modality in study of the speech mechanism. Using proper equipment and techniques, a variety of important speech problems may be evaluated without excessive radiation exposure.

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These observations are derived from a continuing study of normal and abnormal physiologic mechanisms of the pharyngeal region. Since 1954, various aspects of this project have been under sponsorship of the National Foundation (formerly the National Foundation for Infantile Paralysis), the United Cerebral Palsy Research and Educational Foundation, Incorporated, and the Public Health Service.

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XERORADIOGRAPHY OF THE BREAST*

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ROENTGENOGRAPHY of the breast has been used extensively by several groups in this country with good results, notably by Gershon-Cohen *et al.*[1–6] Several other groups[7,8] have also reported on this method of examination. Reports on its diagnostic accuracy show that while it is quite satisfactory, it is greatly dependent upon the technique used and upon the experience of the radiologist. High quality films are imperative and somewhat difficult to achieve. With these facts in mind we attempted to make xeroradiographs of normal breasts in order to determine whether this method had advantages over conventional roentgenography.

METHOD

Xeroradiography[9,10] is a method in which a selenium coated and charged metal plate is used instead of a film. Roentgen rays, according to their intensity, cause a varying run-off of the charge on the area of the plate which they strike. After exposure, the plate is dusted with charged calcium carbonate powder. An image similar to that seen on conventional roentgenographic film is produced. The entire processing is dry and no darkroom is necessary. The image may be preserved by transferring it to paper by an adhesive technique or by photographing it. The resolving power of the xeroradiographic plate exceeds that of the conventional roentgenographic film. It has been shown in tests with wire mesh that using standard roentgenographic film in a cardboard holder, one begins to lose detail with a mesh of 180 lines to the inch, while a mesh of 1,200 lines to the inch can be recorded on a xeroradiographic plate. Moreover, the wide ranges of contrast seen on roentgenographic film are not seen on the xeroradiograph. For instance, in a xeroradiograph of the skull, the skin and subcutaneous tissues are clearly seen as well as the bone. Similarly, there is little difference in detail using moderate ranges of kilovoltage and mas. It can therefore be seen that an exact technique is not necessary in order to obtain an adequate xeroradiograph for interpretation.

MATERIAL

Thirteen female patients, ranging in age from eighteen to fifty-eight years were selected at random from the Outpatient Clinic of this hospital. They were seen for various disorders but none had symptoms referable to their breasts. All were examined by one of us (R.S-U) and then xeroradiographs were made. Lateral views of the breast were obtained with the patients in the decubitus position. The following factors were used: 50 kv., 100 ma., one quarter to one half second exposure, and 30 inch distance. The plates were examined immediately after dusting with calcium carbonate powder and then were photographed. Some detail was lost in this process.

DISCUSSION

In all, 22 breasts of 13 patients were examined. The results were as follows: good

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The views expressed are those of the authors and not necessarily those of the Department of the Air Force.
The equipment used in this study was generously provided by the X-Ray Division of the General Electric Company.
† Chief Resident, Department of Radiology, St. Vincent's Hospital.
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visualization of the breast structures, 14; fair visualization, 6; and poor visualization, 2. These figures are more significant when one considers that this was our first experience with soft tissue radiography of the breast.

Figure 1 is a composite drawing of the structures that may be seen in the xeroradiograph. As with conventional soft tissue roentgenography of the breast, the parenchymal structures are more easily identified when outlined by the greater fatty content of the breast with increasing age. However, even in the younger patients, the ducts and trabeculae are well seen. The chest wall itself is also clearly demarcated. The skin and subcutaneous tissue are not overpenetrated as occurs more readily in conventional soft tissue roentgenography. Vessels coursing in the subcutaneous tissue stand out distinctly from the breast parenchyma itself. On several of the xeroradiographs, the reticular appearance of the subcutaneous fat was seen.

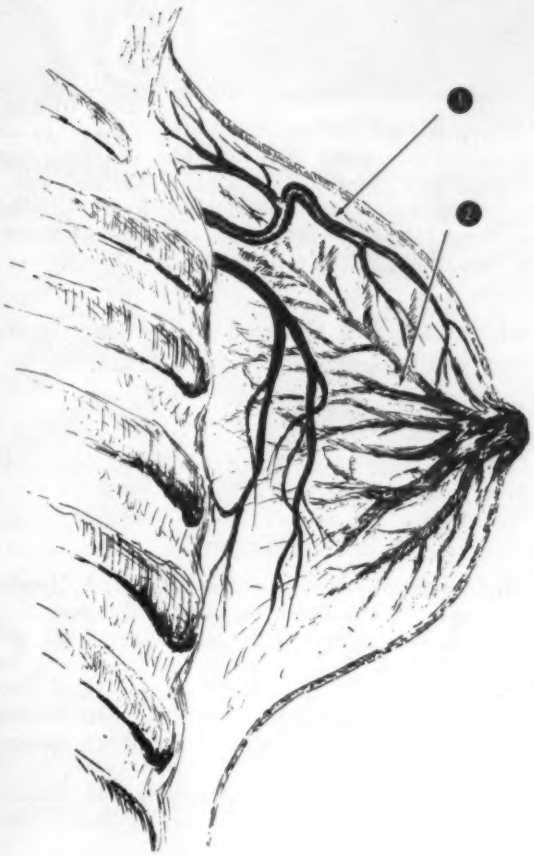

Fig. 1. Composite drawing of normal breasts as shown by xeroradiography. (1) Blood vessel. (2) Duct.

Fig. 2. The right breast of a twenty-nine year old nulligravida. The soft tissues are well visualized. The ribs, lung and diaphragm can be clearly seen.

Figures 2 and 3 show the right and left breasts, respectively, of a twenty-nine year old nulligravida who clinically had "slight fibronodular disease around the nipples." On the xeroradiographs, no evidence of any abnormality is seen in the breast parenchyma. The trabeculae and ducts are well visualized, especially in the area immediately below the nipples. Detail is distinct as deep as the chest wall. The skin and subcutaneous tissues are clearly seen as are the subcutaneous vessels in the superior portion of the breast.

The postpartum breast of an eighteen year old multipara is illustrated in Figure

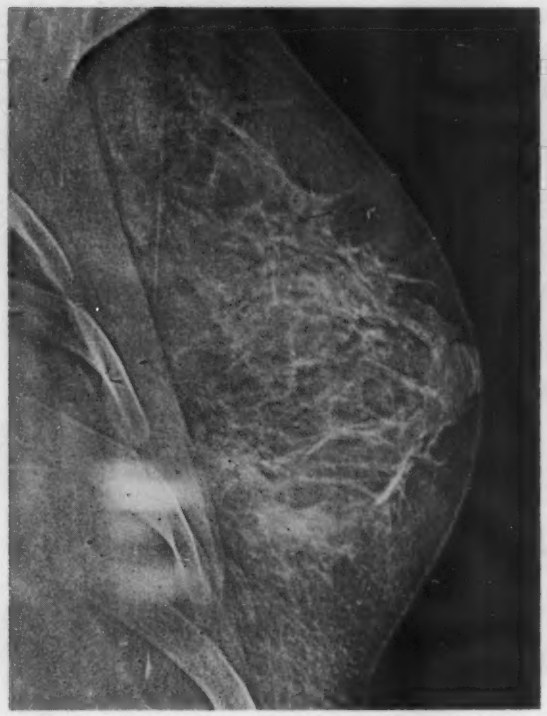

FIG. 3. An enlarged view of the left breast of the same patient shown in Figure 2. Detail is sharp in the subcutaneous tissues, the parenchyma, and behind the breast itself.

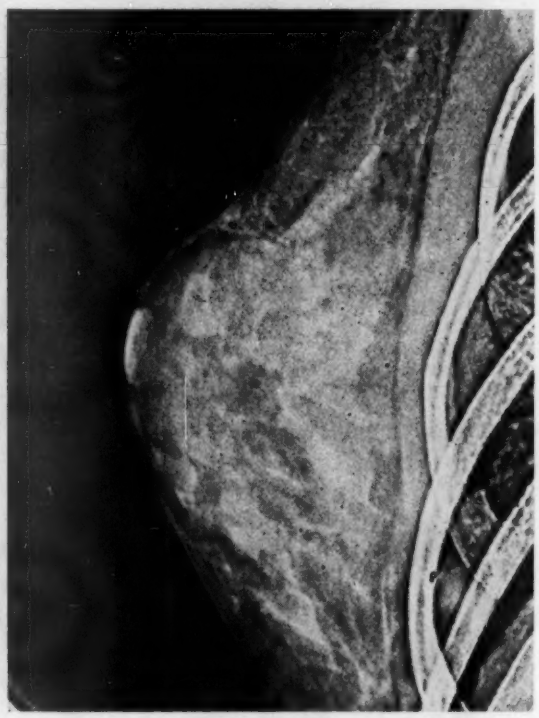

FIG. 4. An enlarged view of the right breast of an eighteen year old multipara who was forty-five days post partum. Individual glands and ducts can be made out with difficulty. However, detail is much sharper than with conventional roentgenography in this type of breast.

4. The dilated edematous ducts and trabeculae are shown. In this type of breast, conventional soft tissue roentgenography will often show an almost homogeneous density.

SUMMARY

A new method for soft tissue breast radiography is presented which we believe has advantages over the conventional soft tissue roentgenography. Because the resolving power of the xeroradiographic plate is greater than that of the conventional roentgenographic film, greater image detail is achieved. Similarly, there is not found the wide range of contrast seen on conventional roentgenographic films so that all the structures from the skin to the chest wall and ribs may be well visualized. We feel that this method deserves further trial in the evaluation of breast lesions, especially those that are adjacent to the chest wall, where detail is apt to be poor when conventional roentgenography is used.

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TECHNICAL IMPROVEMENTS IN BREAST ROENTGENOGRAPHY*

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ACCEPTANCE of routine roentgenography of breast diseases has been retarded partially by technical difficulties. Some of these can be overcome easily with experience; others can be solved by the use of special apparatus and film. Our present methods of examination might be of interest to those giving this subject serious attention.

Routine roentgen-ray study is done with exposures in the cephalocaudad and lateral projections of the breast. The cephalocaudad roentgenogram is obtained with the patient erect and the lateral roentgenogram with the patient in the lateral decubitus position. Good collimation is achieved with a flat-sided cone which can be pressed against the chest wall.[1,2,3] For optimum contrast, non-screen films, 30 kv. and 100–250 mas. are used with a focal spot of 0.3 mm. at a film target distance of 35 cm. The variation in the mas. factor depends on the size and solidity of the breast. A young compact virginal breast of equal size with a multiparous fatty breast will require doubling or tripling of the mas. factor. A large compact breast will require also an increase in kilovoltage, whereas an atrophic breast of equal size with much fatty infiltration will require reduction in both the kilovoltage and mas. factors. These modifications of exposure factors are learned with experience.

Most radiologists will come to prefer roentgenograms slightly overexposed, viewing them in brighter light than is ordinarily used for routine study. Finally, it has been found useful to place a small lead shot on scotch tape over any area of pain or a lesion which is palpable. If a lesion merits particular attention, additional spot compression roentgenograms are made in any projection which will bring the suspicious area closest to the film.

The pyramidal or globular contours of the breast interfere with obtaining uniformly good contrast of all the structures on a single film. If the factors are suited best for good contrast of the deep structures, then the superficial portions of the breast are overexposed. If the anterior segment of the breast is shown in good contrast by a given set of exposure factors, then the deeper portions near the chest wall will be underexposed. To overcome these difficulties, simultaneous exposure is made of two non-screen films, interposing a thin foil of aluminum 0.5 mm. in thickness. The upper film then reveals good contrast of the thicker portions of the breast and the film covered by the aluminum foil reveals good contrast of the anterior portions of the breast. Two films thus are obtained with one exposure, totaling no more than 0.5 r to the skin (Fig. 1, *A* and *B*).

The poor quality of illustrations appearing in many reports also retards appreciation of breast roentgenography. Log-Etronic reproduction overcomes some difficulties, but the use of Ferrania direct duplicating orthochromatic film has been found more satisfactory. This film is specially designed to obtain a direct positive copy from the original, using either arc, fluorescent or ordinary electric light bulbs. Processing is the same as with ordinary non-screen roentgen-ray film (Fig. 2, *A* and *B*).* It is hoped that, in the future, illustrations by this method will do more justice to representation of breast diseases.

* This method was first suggested to the author by Dr. W. J. Jansen of the University of Louvain. Film was supplied by Ferrania, Società per Azioni, Milan, Italy.

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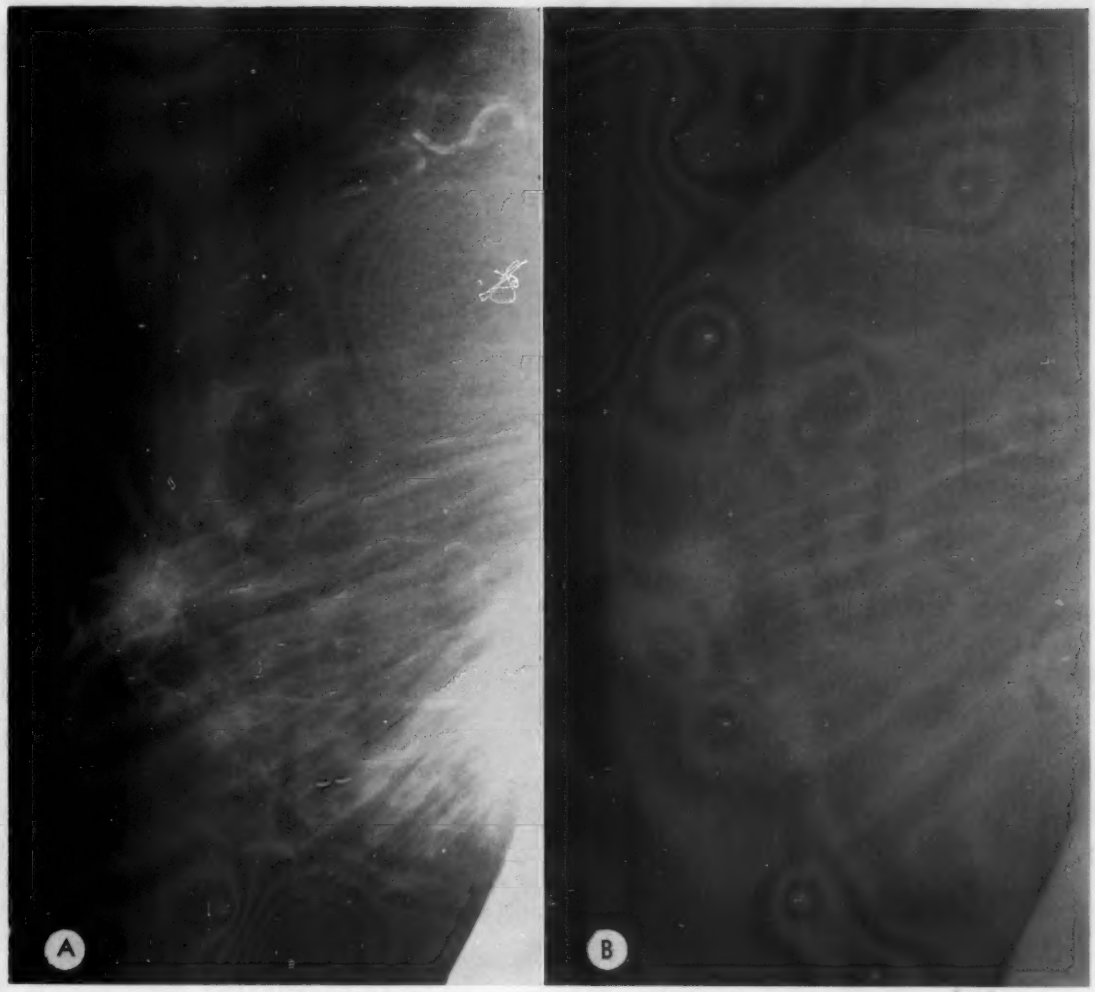

FIG. 1. (*A*) Subareolar carcinoma. The structural components of the deeper portions of the breast stand out in good contrast, but the nipple and the thickened skin in the areolar area are lost to view in the overexposed portions of this film. (*B*) The size and shape of the subareolar lesion, the thickened skin and nipple are seen better in this exposure made through a cover of aluminum foil 0.5 mm. thick, interposed between this film and the one in *A*.

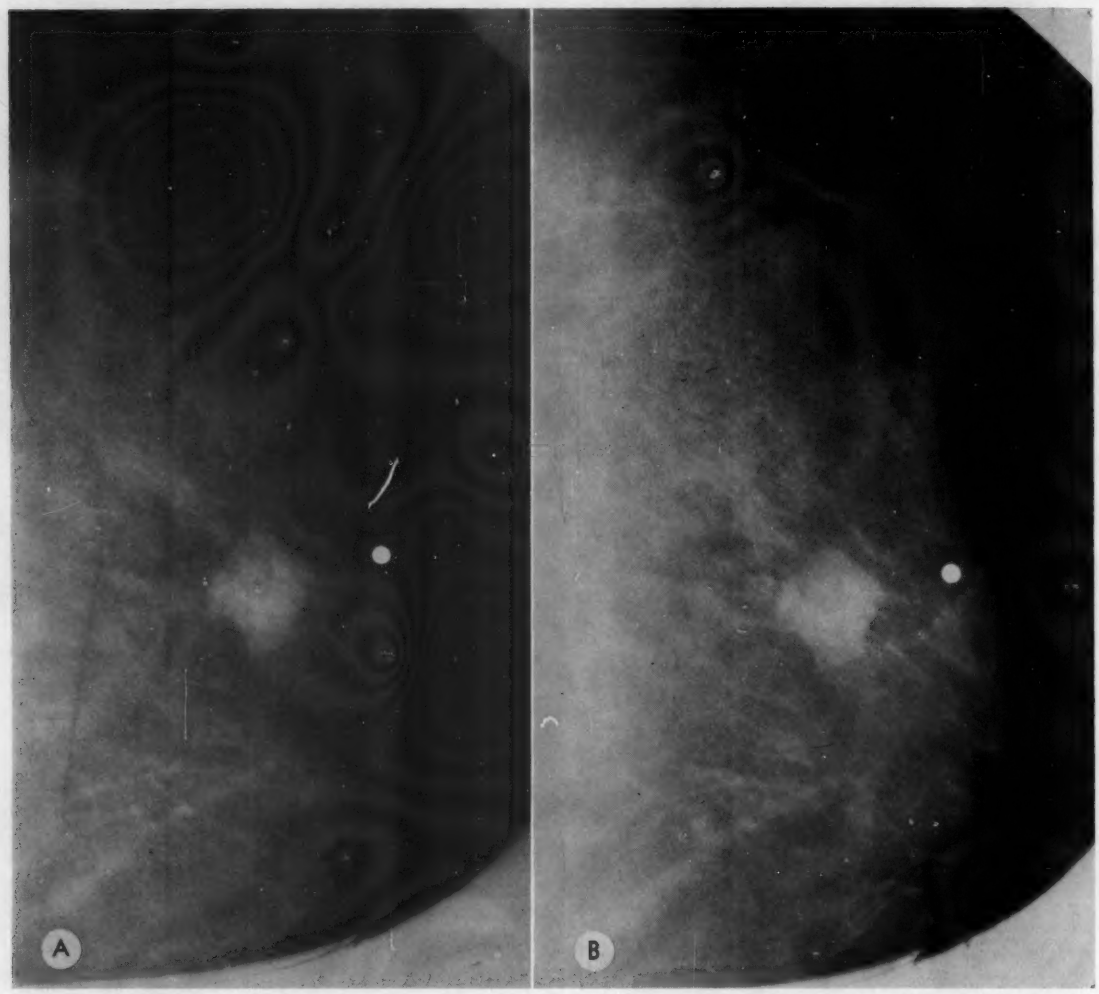

FIG. 2. (*A*) Carcinoma and structural details of a breast as revealed on a non-screen film reproduced without dodging or other artificial alteration. (*B*) Same as *A* reproduced from the original on a direct duplicating orthochromatic film revealing many additional structural details.

Breast roentgenography thus requires strict adherence to a good technical regimen and it is hoped that the suggestions in this report will be helpful to those pursuing this subject.

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ROENTGEN MANIFESTATIONS OF HODGKIN'S DISEASE INVOLVING THE URINARY TRACT*

REPORT OF A CASE WITH URETERAL AND BLADDER LOCALIZATIONS

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URINARY tract localization of Hodgkin's disease is rare. In his large series of cases collected over a period of three decades, in Geneva, Switzerland, Gilbert[5] found no urinary manifestations. In 39 cases of lymphoblastoma involving the urinary tract, Barney *et al.*[1] found only 2 cases of Hodgkin's disease. Boyd[3] reported 8 cases of Hodgkin's disease out of 41 cases of lymphomatous involvement of the genitourinary tract. Watson *et al.*[14] based their study on 1,073 patients with lymphoblastoma. In this series 383 had Hodgkin's disease and among them only 5 showed genitourinary manifestations, none in the bladder. The scarcity of vesical localization of Hodgkin's disease prompted us to report the following case.

REPORT OF A CASE

S.P., a thirty-four year old white male, was well until May, 1954. At that time he noticed a small painless lump on the right side of the neck. On admission to St. Mary's Hospital, except for the cervical tumor, the physical examination was irrelevant. The chest roentgenogram was normal, the Wassermann test negative and blood morphology normal.

The surgeon performing the biopsy noted an "enlarged chain of soft rubbery glands adherent but not firmly matted together" along the posterior border of the right sternocleidomastoid muscle.

Histologic sections revealed that the architecture was markedly disturbed by an infiltration of cells which showed moderate variation in size and shape. In some areas remnants of lymphoid follicles could be seen. The pattern throughout all the lymph nodes was variable and in some regions large numbers of eosinophils predominated the picture. Occasional giant cells were seen. The diagnosis was Hodgkin's disease of lymph nodes.

The patient was referred to the Radiation Therapy Department for treatment of the involved right cervical and supraclavicular lymph nodes. This lymphadenopathy regressed following administration of medium doses of roentgen therapy.

In October, 1955, the patient experienced a sudden sharp pain in the right lumbar region. Soon the pain became persistent and was accompanied by an irregular spiky temperature elevation. Intravenous pyelography disclosed right hydronephrosis with dilatation and medial displacement of the right ureter. Cystoscopy revealed normal appearance of the bladder. The retrograde pyelogram (Fig. 1) confirmed the right hydronephrosis. There was marked narrowing of the distal few centimeters of the right ureter, which showed an irregular scallopped outline. Multiple urinalyses gave specific gravities varying between 1.001 and 1.025. The sedimentation rate was 76 mm. in one hour. The white blood cell count was 5,500 with the following differential: eosinophils 3 per cent, polymorphonuclears 67 per cent, lymphocytes 27 per cent and monocytes 3 per cent. The roentgenograms of the chest and lumbar spine were normal.

The patient's symptoms and the pyelographic changes were attributed to pressure on the right ureter by enlarged granulomatous lymph nodes with possible direct invasion of the distal few centimeters. The patient was subjected to roentgen therapy which was carried out from December 27, 1955 to January 13, 1956. Two anterior and two posterior opposing portals, each 110 cm.^{2} covering the right flank and iliac fossa, were irradiated using the following factors: 260 kv. peak, 15 ma. Thoraeus 1 filter, 1.9 mm. Cu half value layer, 50 cm. focal skin distance. The estimated depth dose delivered over a period of eighteen days was

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FIG. 1. Right retrograde pyelogram. Dilatation and medial displacement of the ureter. The distal pelvic segment is markedly narrowed and irregular in outline.

1,100 r. The patient supported poorly the treatment, which was discontinued prematurely. The symptoms subsided somewhat, but four months later a dull back pain reappeared and the temperature rose again to 102°F. Repeat pyelographic study disclosed nonfunctioning of the right kidney. The left upper collecting system and the bladder were morphologically normal. The urinalyses were quite similar to the previous ones. The blood picture did not change significantly and no anemia was present. Cystoscopy and a retrograde pyelogram (Fig. 2) showed correction of the right ureteral displacement, but the right hydronephrosis persisted. The dilated right ureter tapered off in the pelvic segment. The last few centimeters remained markedly narrowed but the outline appeared smooth. In an attempt to relieve the ureteral obstruction, a new series of roentgen treatments was carried out from May 22 to June 6, 1956. Using the same factors as previously with the exception of the filter which was changed to Thoraeus II, a tumor dose of 1,400 r was delivered in twelve treatments over a period of sixteen days through two opposing portals centered on the narrowed ureteral portion. The subsequent pyelograms showed an excellent response to this therapy. The right kidney recovered its excretory function, the ureter became again normally patent and the hydronephrosis disappeared.

After a period of relative well-being, the systemic symptoms recurred in March, 1957. The

FIG. 2. Right retrograde pyelogram after first roentgen therapy series. Persistence of hydronephrosis. Correction of the medial displacement of the ureter. The distal segment remains narrow but its outline became smooth.

Fig. 3. Intravenous pyelogram after irradiation of the distal segment of the right ureter. Disappearance of right hydronephrosis. The lower pelvic portion of the right ureter shows now normal caliber and regular outline. Fullness of the left ureter. Filling defect in the bladder.

patient complained of general malaise, weight loss, irregular temperature with a spiky curve, and low back pain radiating to the left leg, which became edematous. A control pyelogram (Fig. 3) showed persistence of the favorable results on the right side obtained by previous irradiation. The right upper collecting system was of fairly normal appearance and the distal ureteral segment was of normal caliber and smoothly defined. There was, however, some fullness of the left ureter present and a large filling defect appeared in the left part of the vesical dome. This filling defect (Fig. 4) measured 3.2 cm. in the long axis and was sharply, although irregularly, outlined. Cystoscopy on April 5, 1957 revealed a mass on the left side of the wall, above and lateral to the left ureteral orifice, protruding into the bladder. The appearance was that of a cluster of frog's eggs stuck together. The mass did not bleed. The area corresponded to the filling defect noted

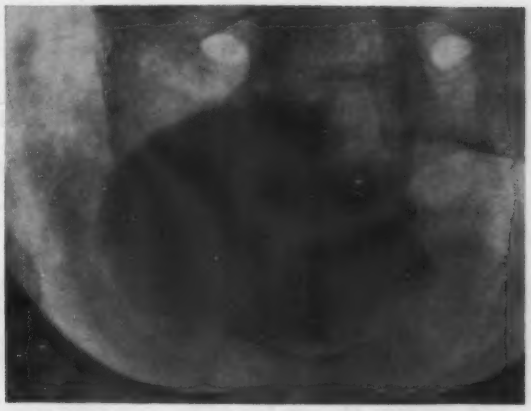

Fig. 4. Urinary bladder with a large, irregular filling defect caused by lymphogranulomatous involvement.

on the roentgenogram (Fig. 4). A biopsy of this mass (Fig. 5) showed that the edematous mucosa was covered by flattened transitional epithelium. The lamina propria and the muscle coat were involved by a pleomorphic infiltrate consisting of reticulum cells and occasional eosinophils. Multinucleated cells were present and some of the reticulum cells were large and showed hyperchromatic bi- and trilobed nuclei. In one section these pleomorphic cells infiltrated

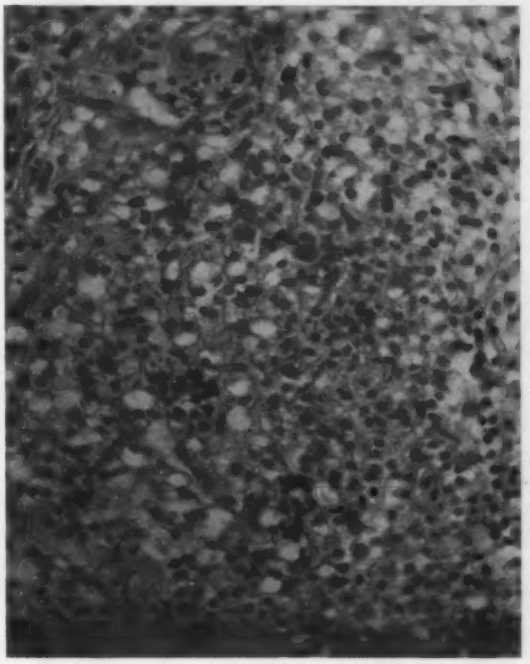

Fig. 5. Photomicrograph of biopsy of abnormal area noted in the bladder. A pleomorphic infiltrate has replaced the bladder musculature. H & E×235.

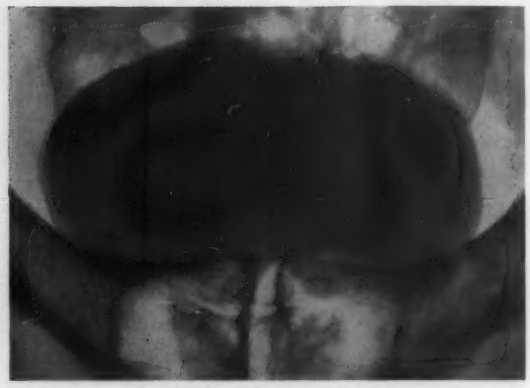

FIG. 6. Urinary bladder twenty-six days after completion of irradiation. The filling defect has disappeared.

through to the serosa. The diagnosis was consistent with Hodgkin's disease of the bladder wall.

Roentgen therapy of the bladder tumor was carried out from April 29 to May 24, 1957, and consisted of crossfiring of the mass through two anterior and two posterior portals to an estimated tumor dose of 2,400 r, delivered in 19 treatments over twenty-six days. Other factors were the same as in the first series. The swelling of the left leg subsided rapidly. A pyelogram taken on the twenty-sixth day after completion of therapy (Fig. 6.) showed disappearance of the vesical filling defect. However, the patient's health continued to deteriorate rapidly in spite of cortisone, nitrogen mustard and supportive therapy. He became cachectic and obstructive jaundice, ascites, hydrothorax, melena and psychic manifestations developed. The temperature rose to 104°F. and the patient expired on September 3, 1957.

The pertinent autopsy findings were (S. 57–607) Hodgkin's disease with involvement of the lungs, spleen, liver, bladder and lymph nodes and obstructive jaundice secondary to compression of the common bile duct by the involved lymph nodes. In the bladder there was a 1.3 cm. in diameter raised pedunculated mass on the mucosal surface. This was located on the posterior wall just to the left and above the trigone. The ureteral orifices were patent and the ureters not dilated. Microscopic sections of the involved area of the bladder showed the mucosa desquamated. The lamina propria exhibited marked fibrosis and scattered through it were masses of eosinophilic homogeneous material with focal calcification. The muscle coat was preserved but fibrosis and focal calcification were present in the serosa. No recognizable viable tumor tissue was found.

DISCUSSION

The urinary organs most frequently involved in Hodgkin's disease are the kidneys. Cases have been reported by Barney *et al.*,[1] Boyd,[3] Jackson and Parker,[7] Papillon *et al.*,[12] Sanseverino[13] and Watson *et al.*[14] In the series reported by Healy *et al.*[6] the renal involvement amounted to 5.5 per cent of cases. The kidney may be displaced by neighboring granulomatous masses or may be infiltrated. The infiltration may be either diffuse or localized with granulomatous masses causing destruction and pressure atrophy of the renal parenchyma. One such case with clinical manifestation of nephrotic syndrome was reported by Papillon *et al.*[12]

Kirkassian (quoted in reference[12]) studied 55 cases of secondary tumoral involvement of the ureters. In his series 3 cases had Hodgkin's disease. The usual roentgen manifestations of ureteral involvement in Hodgkin's disease are displacement and/or obstruction of the ureter. This was true in our case as well as in others reported.[2,3,4,14] In rare instances the wall of the ureter may be infiltrated by the tumor and Papillon *et al.*[12] claim that one of their cases belongs to this category although no pathologic confirmation of it is given.

Involvement of the urinary bladder in lymphoblastoma is extremely rare.[14] Cases of lymphosarcoma are reported more frequently than of Hodgkin's disease.[8,9,11] Mellicow[11] reviewed 2,500 specimens and biopsies from 1,614 cases of urinary bladder tumors and found only one case of Hodgkin's disease. Barney *et al.*[1] quote one case reported by Szenes in which the bladder, uterus, ovaries and tubes were involved. In the classic monograph of Jackson and Parker,[7] 2 cases of bladder involvement are reported, one in a group of 63 cases of Hodgkin's granuloma and another in 32 autopsies of Hodgkin's sarcoma. Healy *et al.*[6] found one bladder involvement in 216

cases of Hodgkin's disease, roughly 0.5 per cent. To our knowledge the case here reported is the sixth one.

According to Watson *et al.*[14] there are four types of lymphoblastoma involvement of the urinary bladder: (1) circumscribed single or multiple foci; (2) direct invasion of the bladder by encompassing perivesical masses; (3) extension from prostate; and (4) mucosal and submucosal hemorrhages.

Our case belongs to the first type. The tumor consisted of a large single mass having the appearance of a cluster of frog's eggs. The tumor did not bleed, was clinically silent and it was discovered roentgenographically. The roentgen diagnosis was made possible by the history of the patient and was confirmed by histologic studies. It is interesting to note that a cystoscopy seven months prior to the discovery of the vesical tumor was negative. The new growth developed and reached its size in this interval.

The pronounced radiosensitivity of Hodgkin's disease is known and the urinary manifestations are no exceptions. In the reported case correction of the displacement and obstruction of one ureter was achieved with two series of roentgen therapy. The vesical tumor was eradicated grossly and histologically, after a tissue dose of 2,400 r was administered.

SUMMARY

1. One case of Hodgkin's disease involving the ureter and the urinary bladder is reported.

2. A review of the available literature is given.

3. The value of radiation therapy is emphasized.

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CUTIS HYPERELASTICA, THE EHLERS-DANLOS SYNDROME

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THE cutis hyperelastica syndrome was described by Ehlers and Danlos and is commonly associated with their names.[1] Initially, four features of the syndrome were distinguished: (1) hyperelasticity of the skin; (2) fragility of the skin and blood vessels; (3) hypermobility of the joints; and (4) subcutaneous nodules overlying bony prominences. Later, Weber and Aitken[8] added a fifth feature, the presence of small subcutaneous "spherules."

Due to the obvious abnormalities of the skin, most cases of this disease have been reported in children.[1,5] Other patients have been diagnosed at later stages because of less prominent features of the syndrome such as subcutaneous hemorrhages, poor healing after trauma, and dislocation of joints.[2]

The following case illustrates the roentgenologic findings in this syndrome and several of the complicating factors which make its medical management difficult.

REPORT OF A CASE

R.S., a thirty-five year old white man, was admitted to the Veterans Administration Hospital, Philadelphia, Pennsylvania, on August 25, 1953, with a history of intermittent swelling of the left lower leg and ankle for about twenty-four years. For eight years prior to admission he had been unable to work because of leg cramps and ankle swelling. For several years he had noticed easy bruising. On the morning of admission he had noted a red, tender nodule on the anterior surface of the left lower leg.

Physical examination revealed a well nourished white man in no distress. The significant physical findings included unusually small hands and feet and laxity of the abdominal musculature. The left ankle was diffusely enlarged and tender. There was a red, tender nodule measuring 4 cm. in diameter on the anterolateral aspect of the left ankle. Multiple "cigarette paper" scars were noted over the extremities, especially the anterior surface of the legs. There was moderate hyperextensibility of the finger joints. Several ecchymotic areas were seen on the right foot.

Laboratory studies, including complete hematologic examinations, were within normal limits. Roentgenologic examination revealed multiple, small calcific densities in the soft tissues of both legs (Fig. 1 and 2) and in the right thigh near the greater trochanter of the femur. These densities showed a ring-like outer shell with a central relative radiolucence. Additional roentgen examination of the gastrointestinal and urinary systems showed no abnormality.

A biopsy of the nodule and the adjacent muscle was performed. Microscopic examination revealed a hematoma and normal muscle. The small surgical incision healed without incident. Because of the skin changes, hyperflexibility of the joints and the roentgenologic findings, a diagnosis of Ehlers-Danlos syndrome was made. The patient was discharged on October 9, 1953, in an asymptomatic condition.

During the next twenty-one months the patient was re-admitted to the hospital on three occasions because of subcutaneous hemorrhages, herpes zoster and an episode of acute diverticulitis which responded to conservative management.

On July 18, 1955, he entered the hospital complaining of severe abdominal pain of several hours' duration. Physical examination revealed a temperature of 102°F., pulse of 130 and moderate dehydration. There was tenderness and spasm in the right lower quadrant of the abdomen and rebound tenderness referable to this area. Because of the possibility of acute appendicitis, an exploratory laparotomy was performed on the day of admission. A severe regional enteritis involving most of the small intestine was encountered. There was marked elasticity and friability of all tissues, and a definite tendency to hemorrhage from capillary sites. Postoperatively, the patient's course was stormy. On July 21, 1955, there was dehiscence

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Fig. 1. Multiple, calcific cystic lesions in the soft tissue of the legs, characteristic of the Ehlers-Danlos syndrome.

of the abdominal wound. The patient developed a nonobstructive ileus, persistent high fever and severe gastrointestinal hemorrhage. Several days later a rectal prolapse occurred. In spite, of transfusions and supportive therapy, a downhill course continued. On August 2, 1955, roentgen examination showed a large pneumoperitoneum and a decrease in the amount of intraintestinal air. The patient expired on August 4, 1955.

DISCUSSION

The presence of subcutaneous "spherules" in Ehlers-Danlos syndrome frequently permits a roentgen diagnosis to be made. These small cystic lesions are said to contain an oily substance and to represent an abnormal type of fatty growth.[8] It has also been said that the "spherules" result from the avascular degeneration of fatty tissue. The walls of the cysts are fibrous and contain varying amounts of calcium.[5,8] The roentgenographic characteristics depend upon the amount of calcific material in the cyst wall. These cystic lesions may range in size from 2 to 15 mm. in diameter. The typical small nodules in the present case demonstrate an outer ring-like shell with a central area of relative radiolucence. These cysts have been found chiefly in the extremities, especially the lower limbs. A secondary roentgen manifestation, not present in this case, is subluxation of joints due to the extreme hyperextensibility of the periarticular tissues. Advanced traumatic arthritis frequently follows as a

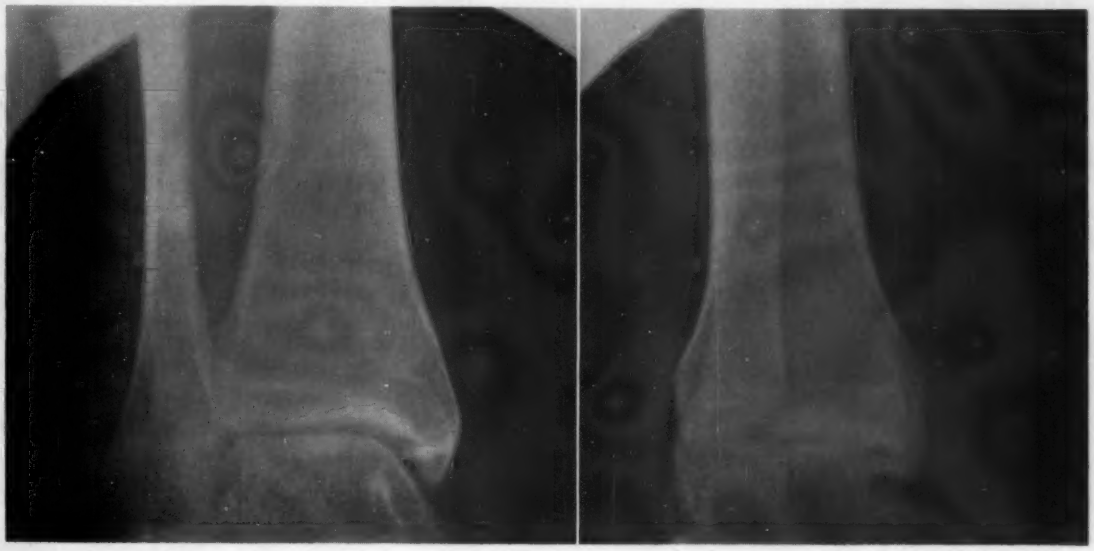

Fig. 2. Similar lesions in the soft tissue of the right ankle.

complication. The larger nodules, which may be present over bony prominences, do not calcify and have been found to consist mainly of fibrous scar tissue. These nodules are believed to result from trauma.

The differential roentgen diagnosis includes several other uncommon conditions. Angiomatous phleboliths are quite similar in appearance, but usually not subcutaneous in location, nor are they ordinarily as widespread and numerous as the calcifications in the Ehlers-Danlos syndrome.[2] The phleboliths may also demonstrate a ring-like calcification with a relatively radiolucent center. Another cause of subcutaneous calcification is cysticercosis. This condition is more common in central Europe, but has been reported with increasing frequency in the United States. In cysticercosis, multiple subcutaneous calcifications are seen, but usually they are long and tapered, measuring about 1×5 mm.[3]

Pathologically, the defect in cutis hyperelastica has been reported as an abnormality in the elastic tissue fibers. Using special staining techniques, the subepidermal framework has been noted to be dense, with marked irregularity of the fibers. Extensive hyperplasia and thickness of the elastic tissue throughout the cutis have been seen. The microscopic diagnosis is referred to as "hyperplastic elastosis."[5]

CONCLUSIONS

The present case is reported since it illustrates the combination of typical roentgen findings unassociated with the usual marked elasticity of the skin. The patient's chief complaints were centered around manifestations of blood vessel fragility, while the hyperelasticity of the skin and hyperextensibility of the joints were only subordinate symptoms. The abdominal disease, diagnosed as regional enteritis, has been reported associated with the Ehlers-Danlos syndrome in several instances. Tissue fragility, poor wound healing and generally poor reaction to operative procedures are characteristic of the syndrome.

It seems probable that more cases of the Ehlers-Danlos syndrome exist undiagnosed and that the incidental roentgen findings may prove a key to the correct diagnosis in these cases. The importance of recognizing this syndrome is emphasized by the complications which occurred in the described case following an exploratory laparotomy.

SUMMARY

The roentgen features of the Ehlers-Danlos syndrome have been reviewed and a case report presented. The important roentgen finding is that of multiple, small, subcutaneous nodules of calcific density, seen particularly over the lower extremities. Another roentgen manifestation, not seen in the present case, is abnormal joint relationships due to hyperextensibility of the periarticular tissues.

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ROENTGEN MANIFESTATIONS IN PROGRESSIVE SYSTEMIC SCLEROSIS (DIFFUSE SCLERODERMA)*

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THE term, "progressive systemic sclerosis," was recommended by Goetz[11] in 1945 to replace the name, scleroderma. This is a good descriptive term which indicates the most important features of this disease; *i.e.*, (1) "Scleroderma is only one of the symptoms and we are dealing with a systemic disease neither solely nor primarily involving the skin."[11] The generally accepted concept, that this is a disease of the connective tissue, is in accord with this name; (2) it is a progressive disease leading to prolonged disability and, commonly, to death of the patient; and (3) the term suggests the most obvious manifestation of the disease, a leathery induration and sclerosis, generally occurring in the skin. The sequence of events is the same in every organ: (a) edema, followed by (b) proliferation of the connective tissue and sclerosis of the collagenous bundles (induration) and finally (c) atrophy.

CLINICAL MANIFESTATIONS OTHER THAN SCLERODERMA

Clinical manifestations of the disease in the heart, lungs, esophagus and small intestine, without obvious involvement of the skin, have been reported from various sources. Lindsay *et al.*,[18] in describing 5 cases of involvement of the esophagus, mentions a case in which the symptoms of dysphagia preceded those of the skin by one and one-half years. Of 9 patients with cardiac involvement reported by Weiss *et al.*,[34] 3 presented cardiac manifestations prior to involvement of the skin. In a review of 27 cases from the literature, Hayman and Hunt[14] found 7 cases where pulmonary symptoms of progressive systemic sclerosis preceded the appearance of cutaneous changes. Abrams *et al.*[1] described, among others, a patient in whom the roentgenologic changes were so prominent that the diagnosis of scleroderma was suggested on the basis of a small intestine roentgenographic study, when no clear-cut clinical diagnosis was apparent. The skin manifestations at that time were minimal and were overshadowed by the gastrointestinal involvement. Piper and Helwig,[27] in a study of 31 autopsied cases from the Armed Forces Institute of Pathology, found 4 instances where pulmonary symptoms were the initial clinical manifestation. In these 4, "scleroderma" of the lungs was observed on pathologic examination. Only 1 of the 31 cases had presented initial cardiac symptoms. Marshall[20] reported on 3 patients who were submitted to surgery on the basis of a diagnosis of intestinal obstruction and in whom no obstruction was found; histologic study of the resected specimens resulted in a diagnosis of "scleroderma." No obvious skin lesion was evident in these patients at the time of surgery. In a recent publication, Herrington[15] submitted the case history of a fifty-five year old farmer who had intermittent, crampy, abdominal pains of six years' duration. At the time of admission, the patient presented the clinical syndrome of small intestinal obstruction. Roentgenologic examination of the upper gastrointestinal tract revealed absence of peristalsis and dilatation of the esophagus; there was marked delay in the passage of barium. The small intestine was dilated and no point of obstruction was

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demonstrable. A diagnosis of scleroderma was suggested. On surgical exploration, a long segment of the jejunum and upper ileum was found to be atonic and flaccid; the bowel wall was thin and grayish white. A resection of the involved segment was performed. Both the gross pathology and the histology indicated scleroderma.

These observations not only emphasize the correctness of Goetz's concept of the disease, but also indicate the important role of the roentgenologist, who may be the first to suggest the diagnosis. A comprehensive review of the subject and the literature was presented by Pugh[28] in 1948 and subsequently by Boyd *et al.*,[3] Meszaros,[21] and Orabona and Albano.[25]

MATERIAL AND INCIDENCE

The purpose of the present study is to review the experiences with progressive systemic sclerosis observed in the D. C. General Hospital during the last sixteen years. Table I summarizes the most improtant data. In 17 patients, histologic proof was obtained; this includes the 1 case in which autopsy was performed. In the remaining 8 patients, either no biopsy was obtained or the result of the biopsy was inconclusive. In these cases we had to rely upon the dermatologic manifestation of the disease. The incidence in females was more than twice as high as in males, which corresponds with the reports in the literature. The disease most commonly occurred between the ages of thirty and sixty years.

Table I

INCIDENCE OF PROGRESSIVE SYSTEMIC SCLEROSIS IN D. C. GENERAL HOSPITAL, 1943–1958

No. of Patients	25
Male	7
Female	18
Histologic Diagnosis (including one autopsy)	17
Clinical Diagnosis Only	8
Age Incidence	
20–30	3
30–40	7
40–50	7
50–60	8

Table II

INCIDENCE OF ORGANS INVOLVED IN PROGRESSIVE SYSTEMIC SCLEROSIS

	No. Examined	No. Involved
Esophagus	17	8 (47%)
Small Bowel	15	3 (20%)
Colon	8	3 (25%)
Heart	25	8 (32%)
Lung	25	7 (28%)
Bone	23	6 (26%)
Bone and Visceral Organs		4
Soft Tissue Calcification	23	4 (17.4%)

The frequency of the different organs involved is shown in Table II. The gastrointestinal tract was the most commonly involved visceral organ. Approximately every second patient had involvement of the esophagus; second and third in frequency were the heart and the lung. Bone changes and soft tissue calcification were the least common manifestations. It has been emphasized[28] that involvement of the visceral organs occurs more commonly in those cases in which acrosclerosis is present. Our experience seems to support this observation. In 6 cases in which destruction of the phalanges was demonstrated roentgenographically, 4 showed involvement of one or more of the visceral organs as well.

ROENTGEN MANIFESTATIONS

HEART

Weiss and his associates[34] first reported "scleroderma heart disease" as a clinical and pathologic entity in 1943. Their study was made on 9 patients and included 2 postmortem examinations. The heart is commonly enlarged; it generally has a triangular shape and there is diminished pulsation at fluoroscopy. These manifestations are similar to those seen in myxedema heart disease and in pericardial effusion. The pulmonary artery segment may be prominent in patients who also have pulmonary hypertension. This is the result of the pulmonary fibrosis and endarteritis

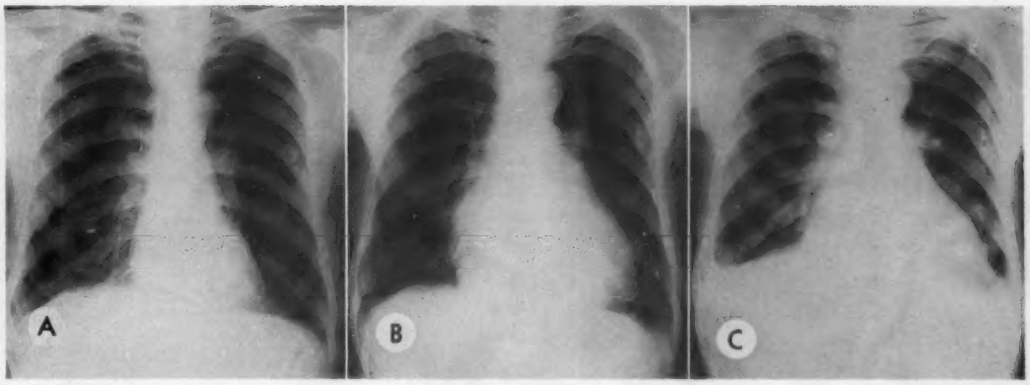

Fig. 1. Scleroderma heart disease in a fifty-nine year old painter, a chronic alcoholic, who had typical scleroderma changes in the hands over a three and one-half year period with recent development of congestive heart failure. (*A*) Roentgenogram of the chest approximately two years after the appearance of scleroderma. Beginning chest pains and shortness of breath. The heart is normal in size and shape. (*B*) One year later increasing decompensation developed. The heart is enlarged both to the right and the left. (*C*) Three and one-half months after *B* there is pleural fluid accumulation in both costophrenic sinuses. The patient died three months after this roentgenogram was made.

associated with progressive systemic sclerosis.[24,32] At autopsy, the cardiac muscles show irregular areas of fibrosis which obviously are not related to any disease of the coronary arteries.[27,34]

In our material, 8 patients (30.8 per cent) showed roentgen manifestations of heart disease. No cases were included in which there was a history or clinical or roentgenologic evidence of congenital, rheumatic or hypertensive heart disease. It is believed that these cases represent scleroderma heart disease though during life a conclusive diagnosis can hardly be made (Fig. 1, *A*, *B* and *C*). Postmortem studies suggest that enlargement of the cardiac silhouette in some cases is probably due to a combination of actual enlargement of the heart and pericardial effusion.

LUNG

Roentgenographic demonstration of pulmonary fibrosis in progressive systemic sclerosis was first reported by Murphy *et al.*[22] in 1941. A review of the pulmonary manifestations was later made by Shuford *et al.*[30] The disease tends to involve the lower lung fields and probably extends gradually in a proximal direction. There may be a diffuse interstitial infiltration and an infiltration intermingled with small cysts. These would present roentgenographic manifestations fairly characteristic of the disease and may be helpful in differentiation from fibrosis of other etiology. Pugh[28] believes that a diagnosis can be made from these roentgen manifestations.

The pathologic changes consist of diffuse fibrosis, thickening of the alveolar membrane and thickening of the wall of the small vessels. The cyst-like lesions represent dissolution of the lung tissue in small areas (from the size of a pin head to 1.5 cm. in diameter) near the pleura, as described by Getzowa[10] and Dostrovsky.[6] In the advanced stage, pneumatoceles may be present, and after rupture of one of these pneumothorax may develop. Such a complication has been reported by Boyd *et al.*;[3] it also happened in 1 of our cases (Fig. 2 *A*). The thickening of the alveolar membrane and of the wall of the capillaries causes difficulty in the gas exchange, which results in two main clinical symptoms, *i.e.*, exertional dyspnea and cyanosis. Impairment of the ventilatory function has a less significant role in the pathologic physiology and symptomatology.

The roentgenologist may be the one who discovers the presence of lung disease and

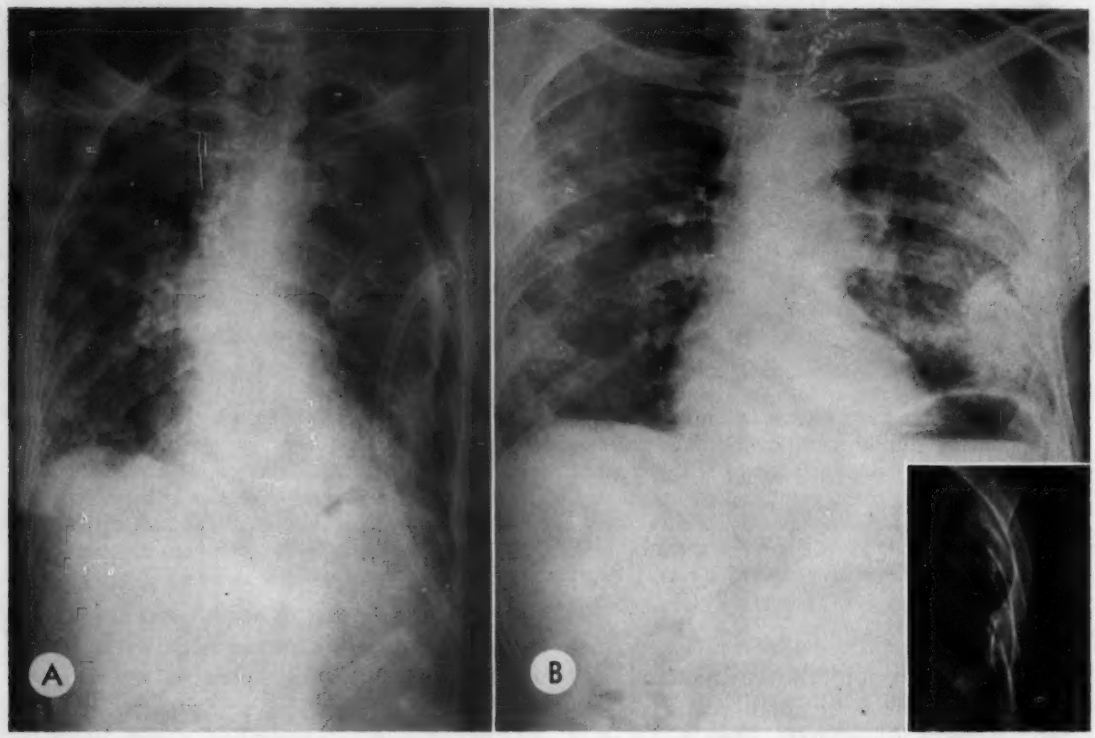

Fig. 2. Pulmonary manifestations in two different patients. (*A*) A fifty-four year old female with advanced scleroderma and complaints of dysphagia. Note the extensive fibrosis and cystic changes in both lungs with spontaneous pneumothorax on the left. (*B*) Roentgenogram demonstrating a probable combination of progressive systemic sclerosis with silicosis in a fifty-five year old miner. Note the extensive pulmonary fibrosis and cystic changes. There is pleural thickening on both sides with linear calcifications within it. *Insert.* Details of calcifications seen in *B*.

he should be aware of the differential diagnostic possibilities. These include sarcoidosis, pneumoconiosis, lymphatic spread of carcinoma, pulmonary fibrosis of Hamman-Rich, histiocytosis, extensive bronchiectasis, and other types of fibrosis. Pleural effusion is rare in progressive systemic sclerosis. Opie[24] quotes 3 cases from the literature where pleural involvement was found by physical examination *in vivo*. In 1 of our cases, bilateral pleural fluid accumulation was demonstrable on roentgenologic examination; it probably represented hydrothorax (Fig. 1 *C*). It has been claimed in the literature that dysphagia, commonly occurring in progressive systemic sclerosis, may result in aspiration pneumonia, and ultimately may be responsible for the diffuse pulmonary fibrosis. This problem was investigated by Mahrer *et al.*[19] in a review of 57 cases of progressive systemic sclerosis where pulmonary fibrosis was present. The study included 16 cases of their own. In these cases, involvement of the esophagus occurred only in 62 per cent. This observation seems to eliminate aspiration pneumonia as a primary cause of the pulmonary fibrosis in progressive systemic sclerosis. Nevertheless, pneumonitis as a result of "spill over" may occur.[17,24]

In 1 of our cases, a combination of silicosis with progressive systemic sclerosis was present (Fig. 2 *B*). This was a fifty-five year old miner with a twenty year history of occupational exposure to silica dust while employed digging tunnels. For two years he had worked on a tunnel through rock which, the patient was told, contained 50 per cent "glass." He had also worked with a cotton gin and in cane sugar

mills. At the time of the examination, the patient had dyspnea on exertion. Purified protein derivative test was positive with 1.5 strength as was the histoplasmin test with 1:100 strength. The patient had the clinical syndrome of progressive systemic sclerosis, which included symptoms and signs of scleroderma as well as changes of the esophagus, small intestine, and colon. There was a history of Raynaud's syndrome and amputation of the lower third of the left arm because of gangrene. A roentgenogram of the chest showed extensive pulmonary fibrosis intermingled with cysts, particularly in the lower lung fields. Both the appearance and distribution of the changes suggested progressive systemic sclerosis and were different from the pulmonary manifestations of silicosis. There were calcified hilar lymph nodes, and the overexposed roentgenograms showed evidence of pleural thickening on the lateral aspects of the thorax. Peculiar calcifications, curved or irregular in direction, were seen mainly in the lateral aspects of the lung fields. The location and distribution of these calcifications suggested that they were in the pleura rather than in the lungs. No pleural or lung biopsy was made.

This unusual calcification deserves particular attention. To this author's knowledge, lung or pleural calcification has not been reported before in progressive systemic sclerosis. Nevertheless, it seems worthwhile to mention in this respect that Goetz[11] did find microscopic calcium salt deposition in the submucosa of the esophagus in an autopsied case of progressive systemic sclerosis. In the group of pneumoconioses, shadows of high density may be present on the roentgenogram if the silica concentration of the inhaled dust is high. Also, after the inhalation of dust containing particles of high atomic number (iron, tin, barium), these particles may accumulate in the lungs and cast nodular shadows of high intensity.[7] Calcifications of eggshell type have been described as more or less typical of silicosis; they may occur in and near hilar lymph nodes. Such an authority in the field of silicosis as Pendergrass[26] states that calcification in silicosis indicates the presence of tuberculosis or histoplasmosis; nevertheless, it has been claimed by Riemer[29] that such calcification may occur without tuberculosis, particularly in slate quarry workers (Davis[5]). The appearance, localization and distribution of these calcifications differ from those of the case under discussion. Extensive pleural calcification, in addition to fine pulmonary fibrosis, is described in silicatosis of talc workers (Siegal *et al.*[31]), occurring in 6.3 per cent of the workers examined. These talc plaques are claimed to involve the visceral pleura. We believe that the calcification in our case is of similar nature, though it is less extensive.

It should also be taken into consideration that the patient's response to dust may be different from that of an average person, if the patient suffers from a collagenous disease. This has been demonstrated in cases of rheumatoid arthritis. Caplan[4] made the important observation that multiple, small, rounded, granulomatous masses occur in the lungs of coal miners who also have rheumatoid arthritis. These masses are different in shape and distribution from the lung changes generally seen in pneumoconiosis. Gough *et al.*[12] demonstrated that these nodules contain necrotic collagen and dust; tuberculosis was present in several of the 16 cases investigated. It is recognized that rheumatoid arthritis is not a joint disease only, but other organs may also be involved. For this reason, it has been suggested that "rheumatoid disease" replace the name of rheumatoid arthritis.[8] Progressive systemic sclerosis is related to rheumatoid arthritis in that both are considered to be members of the collagenous disease group. Caplan's study may illuminate the interesting observations on South African gold miners reported by Erasmus.[9] During his eighteen months' association with the Silicosis Medical Bureau in Johannesburg, he saw 17 cases of scleroderma and suggests that the incidence of this disease in underground mine workers

is more common than in the general population. Those miners who were suffering from scleroderma showed a predominance of pulmonary complications with unusual manifestations (acute onset of the chest symptoms, more marked disability and the relatively common occurrence of pleural and pericardial effusion). Though the relationship between progressive systemic sclerosis and silicosis is not understood, it seems likely that each affects the manifestations of the other in the same way that rheumatoid disease modifies the pulmonary manifestations of pneumoconiosis. We believe that, in the case under discussion, the pleural thickening and the particular calcifications are the result of a combination of silicosis and progressive systemic sclerosis.

GASTROINTESTINAL TRACT

Dilatation and decreased peristaltic activity are the most common abnormalities demonstrated on roentgenologic examination. As a result, the passage of barium through the gastrointestinal tract may be considerably delayed. The essential pathologic changes are thickening of the submucosa by increased fibrous tissue and atrophy of the muscular layer. The first systematic study on roentgen manifestations of the gastrointestinal tract was reported by Hale and Schatzky[13] in 1944.

Involvement of the esophagus is the most common manifestation. It may be demonstrated by roentgenologic examination during that phase of the disease when the patient still has no clinical symptoms on which a diagnosis can be based.[13,25] The wide, atonic esophagus is visualized not only by its barium content but also by air in the lumen with a barium coat on the mucosa (Fig. 3, *A* and *B*). This latter finding is due to the fact that, after the passage of the bolus, the esophagus does not contract as the normal esophagus does, but it remains distended. The absence of the normal progress of the barium can be adequately demonstrated only when the patient is in a horizontal position. In this position, the propulsion of the bolus occurs mainly by the transmitted effect of peristalsis in the upper end of the esophagus, on repeated swallowing.[18] The barium will remain in the esophagus for hours when the patient keeps this position but, in an erect position, the bolus will pass by gravity without delay. This helps to differentiate progressive systemic sclerosis from achalasia (Fig. 3 *C*). We have not seen a narrowing of the distal end of the esophagus (4–5 cm. above the esophageal hiatus) described by Lindsay *et al.*,[18] nor have we observed in this series of cases any esophageal hiatus hernia associated with a short esophagus. Olsen *et al.*[23] report 9 cases of hiatus hernia associated with progressive systemic sclerosis, 6 of them of the short esophagus type.

The stomach in this disease generally shows no striking abnormalities. In 1 of our cases, there was thickening and marked irregularity of the mucosal folds (Fig. 4 *D*).

In the small intestine, the changes are generally localized in the proximal portion. Involvement of the duodenum is common (Fig. 4 *A*). This localization may be one of the differentiating features from the sprue syndrome, which may present similar roentgenologic manifestations. The dilatation in the latter condition, however, tends to be localized in the middle part of the small intestine. The dilatation and the delay in transit time in progressive systemic sclerosis is sometimes very marked (Fig. 4, *B* and *C*) and may reach such a degree that obstruction of the small intestine is considered. Reference has already been made to case reports in which surgery for small intestinal obstruction was performed in such a situation. The most important differential diagnostic point is generally the clinical one; the patient with obstruction of the small intestine is acutely ill; this is usually not the case in progressive systemic sclerosis. If the patient is ambulatory, there may be no significant delay in the motility of the diseased small intestine. For this reason, the patient should be kept in a horizontal position when motility is investigated.

The large bowel may show narrowings and characteristic pouch formations.[1,11,13,17] An important feature of this abnormality is

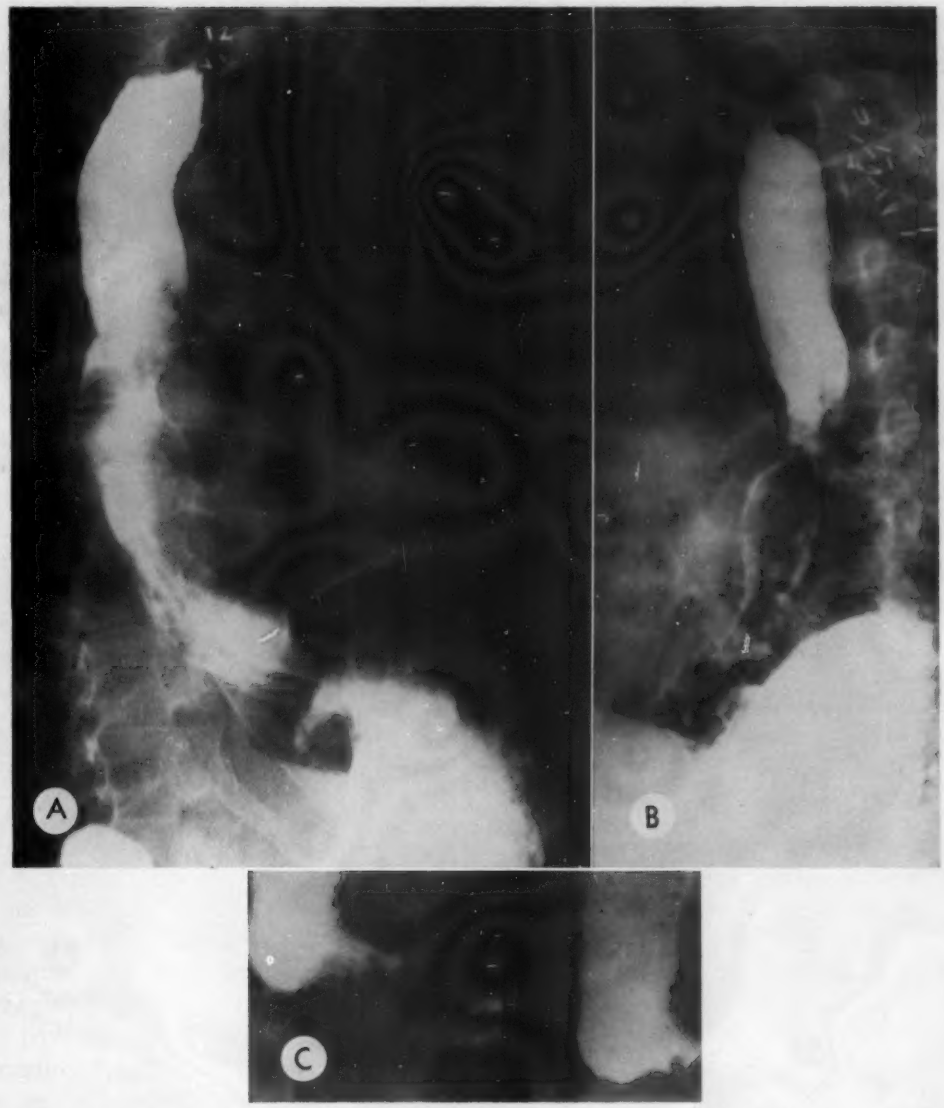

Fig. 3. Esophageal changes in the patient of Figure 2*B*. (*A*) The esophagus is wide and atonic; there is no evidence of peristaltic activity. (*B*) The esophagus did not collapse after the passage of the barium; consequently it became visualized by air. (*C*) At this phase of the examination there is some similarity of the distal end of the esophagus with that seen in achalasia. Nevertheless, there is no obstruction.

the asymmetric location of the pouches in their relationship to the haustrations, as emphasized by Kemp Harper.[17] Haustral contraction may be seen on one side but the pouch formation may not correspond with it on the outer side. The changes generally involve the distal half of the colon (Fig. 4 *C*).

BONES AND JOINTS

Bone abnormalities were found in every fourth case, which is in contrast to the report of Kemp Harper[17] who found a much higher incidence (10 of 11 patients examined). The most common location is the terminal phalanx of the fingers (Fig. 5, *A–C*). The disease generally begins in the distal end of the phalanx; first appearance of the destruction may, however, occur in the middle third of the phalanx, resulting in separation of the distal and proximal portions of the bone, as described by Bársony and Frisch.[2] The distal ends of the radius

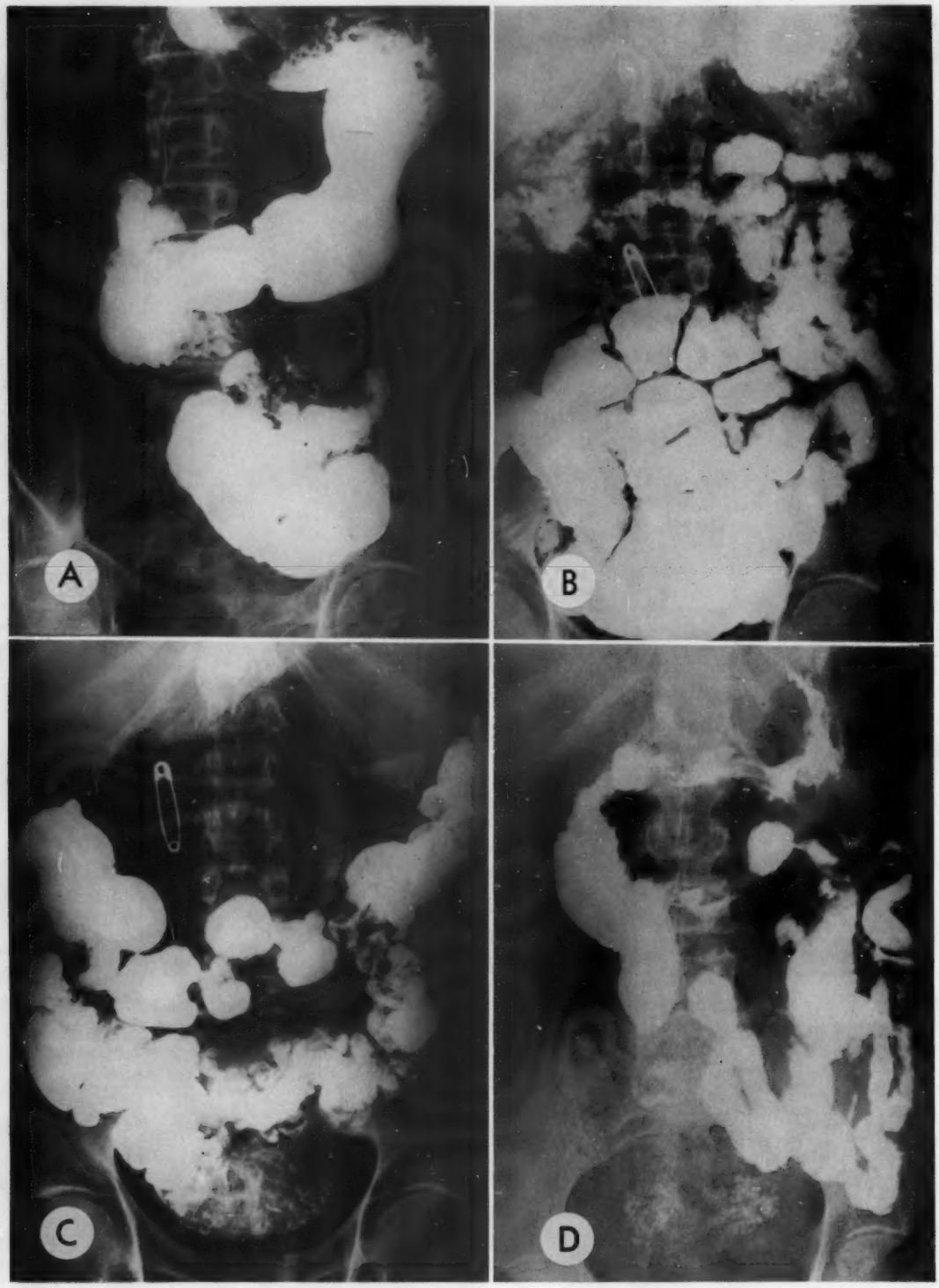

Fig. 4. (*A*, *B* and *C*) Gastrointestinal manifestations in patient of Figure 2*B*. (*A*) One hour after administration of the barium meal. Note retention of barium in the esophagus, its slow progression in the small intestines, the wide duodenum and jejunal loops. (*B*) Six hours later there is still some barium in the esophagus. The small bowel is dilated; the barium column has not yet reached the cecum. (*C*) Twenty-four hours

and ulna may also be involved (Fig. 6 *A*). It is interesting to note that similar changes of the radius and ulna may also occur in rheumatoid arthritis (Fig. 6 *B*). Such a case has been reported by Keats.[16] Involvement of the phalanges of the feet generally shows manifestations identical to those of the fingers, but they are less common. Meszaros[21] reported a case of destruction of the acromion process and of the lateral end of the clavicle.

In the active phase of the disease, an irregular destruction with poorly defined borders may be seen; this becomes smooth during inactive intervals.[2] Progression may stop in one phalanx and, at the same time, may start in another. During inactive intervals, a "reconstruction" takes place with a tendency to approximate the normal shape of the bone (Fig. 5, *A–C*). This commonly results in a conical deformity, with a pointed end of the affected long bone. It should be emphasized that the gradually tapering shape and the smooth contour of the involved bone, in contrast to the general belief, is secondary to the irregular bone destruction in some cases. This author believes that this candle-stick deformity is not specific for progressive systemic sclerosis or any other disease. It is an effort of the living bone to preserve its original shape (tubulation, modelling). The loss of the bony substance is generally considered to be the result of diminished blood supply. This is due to spasm of the small vessels associated with Raynaud's syndrome which occurs in a high percentage of the cases, and to intimal proliferation in the small vessels, which is a common feature of this disease. It has been claimed that the destruction of the bone may represent pressure atrophy, the result of the constrictive action of the skin.[28] The irregular destruction preceding the conical deformity, described and illustrated above, as well as the occurrence of a similar deformity of the radius and ulna are strong evidence against this concept.

The disease commonly begins with joint symptoms, which initially are more frequent than the dermatologic manifestations.[27] The flexion deformity of the fingers and the subluxation and dislocation are the result of the skin disease, the collagenous involvement of the synovial membranes and the bone destruction about the joint.

SOFT TISSUE CALCIFICATION (CALCINOSIS)

Calcinosis associated with progressive systemic sclerosis is usually called Thibierge-Weissenbach syndrome. Calcium salt is deposited in the subcutaneous tissues, particularly in the hands (Fig. 7 *A*). It may also be deposited on the ulnar side of the arm, the elbow and elsewhere. Calcinosis in the soft tissues of the face also has been reported.[21] In our cases, localization in the so-called pressure points was not as prevalent as it is commonly suggested in the literature. We found it on the medial aspect of the arm, in the shoulder and in the breast. In the latter area, the calcification had a worm-like appearance; this calcification probably was located in the lymphatics (Fig. 2 *A*). We have observed circumscribed calcinosis in a patient with rheumatoid arthritis (Fig. 7 *B*). This was a seventy year old woman who had a history, as well as clinical and roentgenographic evidence, of rheumatoid arthritis beginning thirty-four years earlier. The calcium salt deposition was on the ulnar aspect of the right

later the barium retention in the small intestine continues. Note sacculation in the transverse and descending colon.

(*D*) A fifty-four year old female with clinical and histologic diagnosis of scleroderma and complaints of dysphagia, epigastric pain and constipation. Roentgenogram taken two hours after the oral administration of barium shows retention of barium in the esophagus. There is thickening and irregularity of the mucosal folds of the stomach. The passage of barium through the small intestine is considerably delayed. The caliber of the second portion of the duodenum and of the rest of the visualized small intestine is widened. There is no evidence of peristaltic activity in the small intestine.

Fig. 5. A thirty-four year old female with histologically proved scleroderma starting with Raynaud's syndrome four years previously. The distal ends of the terminal phalanges are being progressively destroyed. (*A*) Both hands in 1956. (*B*) Both hands in 1958. (*C*) Right hand in anteroposterior and oblique views in 1959.

hand. During an observation period of two years, this calcification increased somewhat in size. The patient had no evidence of scleroderma.

DENTAL MANIFESTATIONS

Stafne and Austin[33] described uniform thickening of the periodontal membrane in this disease. It may assume 3 or 4 times the thickness of the normal membrane. The involved tooth sits solidly in its socket. The posterior teeth are involved more often than the anterior teeth (Fig. 8). These authors found this abnormality in 7 per cent of 127 cases of acrosclerosis or diffuse scleroderma. The findings are characteristic. On the basis of the dental roentgenogram, they made a tentative diagnosis in 4 cases and this diagnosis was subsequently confirmed on general physical examination.

SUMMARY AND CONCLUSION

During the last sixteen years, 25 cases of progressive systemic sclerosis were observed in the D. C. General Hospital.

Involvement of the heart, lungs, gastrointestinal tract, bones, joints and teeth are discussed and illustrated.

Attention is called to some pathologic manifestations occurring in rheumatoid arthritis which are similar to those of progressive systemic sclerosis. The bone absorption presenting a candle-stick deformity of the distal end of the long bones is preceded, at least in some cases, by irregular destruction of the spongiosa and fragmentation of the cortex. The latter are the specific changes; the candle-stick deformity is considered to be only the result of recon-

Fig. 6. (*A* and *B*) Roentgenograms of the hands of two different patients, demonstrating similar bone manifestations in progressive systemic sclerosis and rheumatoid arthritis. (*A*) A thirty-five year old female with a ten year history of histologically proved scleroderma. There was slight pulmonary fibrosis and esophageal and small intestinal involvement. The roentgenogram shows destruction of the middle third of the terminal phalanx, involving the right second, fourth and fifth fingers. As the result, the proximal and distal portions are separated. Note the conical deformity of the distal ends of both ulnae. (*B*) A patient with advanced rheumatoid arthritis involving the fingers and the wrists. Note deformity of the ulnae similar to that in *A*.

Note the sequence of the destructive processes. *Right middle finger.* 1956: Irregular destruction of the terminal phalanx. 1958: Reconstruction and modelling goes on; the contours are sharper; a few small fragments remain separated. 1959: No change since 1958. *Right fourth finger.* 1956: No obvious bone disease. 1958: Dissolution of the bone substance, mainly involving the spongiosa in the area of the unguinal tuft. 1959: The unguinal tuft grossly disappeared; a small separate fragment is still visible. *Right fifth finger.* 1956: The distal end of the terminal phalanx is absent. The process is inactive (well defined contours, no fragmentation). 1958: No change. 1959: The rest of the phalanx is partly destroyed and fragmented. The oblique roentgenogram shows that the destruction occurred mainly in the spongiosa; the cortex along the whole phalanx begins to be fragmented. The process is similar to that in the middle finger. This sequence of events cannot be explained by pressure atrophy due to constriction of the skin.

Fig. 7. (*A* and *B*) Roentgenograms of the hands of two different patients, demonstrating soft tissue calcification. (*A*) A forty-two year old male with clinical and histologically proved scleroderma, showing soft tissue calcification in both hands. (*B*) A patient with advanced rheumatoid arthritis, showing soft tissue calcification on the ulnar aspect of the left hand.

Fig. 8. A patient with progressive systemic sclerosis, demonstrating marked thickening of the periodontal membrane.

struction. Constriction of the skin as a cause, or one of the causes, of the candlestick deformity is judged to be unlikely. The bone destruction may become active alternatingly in the different fingers. The activity is not necessarily a continuous process but may be interrupted by inactive intervals.

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BONE FORMATION IN LAPAROTOMY SCARS*

ROENTGEN FINDINGS

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HETEROTOPIC bone formation in surgical abdominal scars has been known to pathologists and surgeons for many years. However, the radiologic literature contains very little about the roentgen appearance of these abnormal bone formations which may cast confusing shadows on roentgenograms of the various abdominal structures. In our experience they are often unrecognized or erroneously interpreted. Since only a few roentgenograms illustrating such bone formations in scars of the anterior abdominal wall have appeared anywhere in the medical literature, we feel that it may be worthwhile to present the clinical and roentgen findings in this condition. In this paper 10 patients will be reported in whom bone developed in laparotomy scars and was visible on roentgen studies of the biliary tract, colon, esophagus, gastrointestinal tract, lumbar spine or abdomen.

Ossification of laparotomy scars was originally described by Askanazy,[2] the first example being discussed at the German Society of Pathology in 1900. Since that time approximately 100 cases have been reported. Borrás[6] estimates that about 70 per cent of cases occur in the supra-umbilical region, the rest being infra-umbilical. Roentgenographic verification of the scar ossification was reported first by Bouton[9] in 1926. Silver[30] published the first roentgenogram of such a case in 1931. One of the first detailed studies of ossification in surgical scars was made by Lecène[22] in 1909 at which time he presented 7 cases. In 1911 Bernasconi[4] reported the first instance of ossification in an infra-umbilical scar. By 1948 Abeshouse[1] was able to find only 17 reported cases of infra-umbilical heterotopic ossification following suprapubic operations and added one case of his own.

REPORT OF CASES

CASE I. J.P., a sixty-four year old Negro male, had several episodes of upper gastrointestinal bleeding associated with epigastric complaints. A diagnosis of peptic ulcer was made. On October 15, 1949, a subtotal gastric resection for a prepyloric gastric ulcer was performed through a right pararectus incision. Thereafter the patient was hospitalized on numerous occasions with diagnoses of hypertensive cardiovascular disease, Laënnec's cirrhosis, benign prostatic hypertrophy, alcoholism, and psychophysiologic gastrointestinal reaction.

A gastrointestinal examination in 1958 showed in the epigastrium a peculiar linear structure with several thorn-like projections which emerged from it at acute angles. This was recognized as bone in the nine year old laparotomy scar and a review of the available earlier examinations of the gallbladder and upper gastrointestinal tract indicated that it had been present, but unrecognized, since at least 1956 (Fig. 1 and 2). A roentgenogram in the tangential view revealed the trabecular bone pattern which was not distinguishable in the frontal and oblique views. Examination of the scar which was 20 cm. long disclosed neither local tenderness nor a palpable mass. The patient was unaware of its presence and offered no complaints related to the scar.

CASE II. W.J.H., a sixty-one year old white male, had a posterior gastroenterostomy in 1948 for gastric outlet obstruction secondary to chronic duodenal ulcer. Details of his postoperative course are not available. He was re-hospitalized on November 16, 1957, for complaints of persistent, black, watery diarrhea of six months' duration. There were no symptoms referable to the gastrectomy scar. Liver biopsy revealed metastatic carcinoma of undifferentiated type and undetermined origin. The gastro-

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Fig. 1. Case 1. Posteroanterior roentgenogram of the epigastric area (gastrointestinal series) made seven years after subtotal gastrectomy through right upper pararectus incision. A linear shadow, 6.0×0.2 cm., is superimposed upon the right transverse process of L2 (arrows). It is of different density from the barium in the neighboring small intestine and is in the position of the common bile duct. It is not readily recognizable as bone in this view. On anteroposterior roentgenograms of the abdomen it was not visible at all because of superimposition upon the spine.

intestinal examination showed a normally functioning gastroenterostomy. Heterotopic bone formation was recognized in the right upper abdominal quadrant (Fig. 3). In the posteroanterior view the bone was superimposed on the lumbar vertebrae (Fig. 4). The patient went rapidly downhill and expired. No postmortem examination was obtained.

Case III. R.J.H., a fifty-one year old white male, was hospitalized on July 1, 1955 with complaints of anorexia, weight loss, angina pectoris and exertional dyspnea. A barium enema study revealed a cecal carcinoma which was confirmed at laparotomy on July 12, 1955. A right hemicolectomy and ileo-transverse-colostomy were performed. Postoperatively the patient developed wound dehiscence, mechanical small bowel obstruction and wound infection. A chronic draining sinus developed in the operative scar and has persisted to the present time.

A barium enema study on December 27, 1955, five months after the operation, showed no evidence of bone formation in the scar. On June 21, 1956 the patient was re-examined and a large irregular bone fragment was recognized in the right upper abdominal quadrant (Fig. 5). Roentgenograms in the tangential view proved it to be situated within the abdominal incision. Instillation of iodochloral into the sinus tract and abscess cavity demonstrated no connection between these and the bone (Fig. 6). A roentgenogram in the oblique view showed the bone overlying the right renal shadow (Fig. 7). The patient had no complaints referable to the bony mass, which was nontender on palpation, whereas tenderness was elicited inferiorly at the level of the infected portion of the wound. There was no local discomfort on bending. The interval between operation and detection of the newly formed bone was approximately one year, although it had not formed within the first five months after operation.

Case IV. G.F.V., a seventy-eight year old white male, had a suprapubic prostatectomy for benign prostatic hypertrophy on January 26, 1953. A subtotal gastrectomy through a left

Fig. 2. Case I. Oral cholecystogram in the oblique view shows the bone lying at the right lateral aspect of the fundus of the opacified gallbladder. Its density is very similar to that of the gallbladder.

paramedian incision for a benign, greater curvature, gastric ulcer was performed on May 17, 1955. A drain was placed deep in the abdomen in the region of the duodenum. Postoperatively the patient developed wound dehiscence, wound infection and a ventral hernia. The wound continued to drain through a tract in its superior portion. He was re-hospitalized a month after discharge. At this time a nontender, stony-hard, plum-sized mass was felt just to the left of the midline in the operative scar. The examining physicans considered this mass to be a "calcified hematoma."

Roentgen study of the area was performed on July 27, 1955, approximately nine weeks after operation and disclosed bone formation which was recognized as being newly formed in the laparotomy scar (Fig. 8). A review of the preoperative abdominal roentgenograms confirmed the fact that the bone had not been present before operation. The patient was also noted to have a large left renal calculus. This was followed by pyelonephritis for which a left nephrectomy was done. The heterotopic bone in the gastrectomy scar was not removed. There were no complaints referable to the bony mass which was nontender on palpation. It is noteworthy that bone developed only in the upper rectus scar and not in the suprapubic or nephrectomy scars.

Case v. L.N.R., a thirty-eight year old white male, had been operated upon for a perforated duodenal ulcer in 1954. Shortly afterwards a ventral hernia was noted in the right rectus incision. The hernia gradually became larger and the patient entered the hospital for surgical repair. Within the incisional hernia, just to the left of the midline in the epigastrium and within the previous scar, a firm, mobile, nontender 6×3 cm. mass was palpated. Physicians who examined this patient considered the mass to be either a cartilaginous foreign body or a calcified hematoma. This was presumed to have formed

Fig. 3. Case II. A posterior gastroenterostomy had been performed nine years earlier. Roentgenogram in the left oblique view of the barium filled stomach reveals a semilunar bone fragment, exhibiting cortical and medullary structure, and measuring 7.0×1.0 cm., to the right of L1 and L2. A smaller fragment extends medially from its upper end.

within the one year interval which had elapsed since the operation.

On January 6, 1955, a ventral hernia repair was performed and the mass removed with the old scar. Pathologic examination of the specimen disclosed mature bone (Fig. 9). In this case the heterotopic bone had not been recognized as such on the preoperative roentgenograms nor was there clinical awareness of this entity. Only a chance conversation with the surgeon who had repaired the ventral hernia led to restudy of the case and disclosure of the true nature of the mass which had been palpated in the incision. A review of the barium enema roentgenograms which had been made as part of the preoperative examination showed the heterotopic bone clearly (Fig. 10). Similarly, a preoperative study of the gallbladder had revealed the same previously unrecognized shadow in the plane of the common bile duct (Fig. 11). The actual location of the structure in the anterior wall could have been confirmed on a lateral roentgenogram of the lumbar spine which had also been made preoperatively.

Fig. 4. Case II. Roentgenogram in the posteroanterior view of the upper abdomen shows the bone fragments superimposed on the shadow of the upper lumbar spine (arrows).

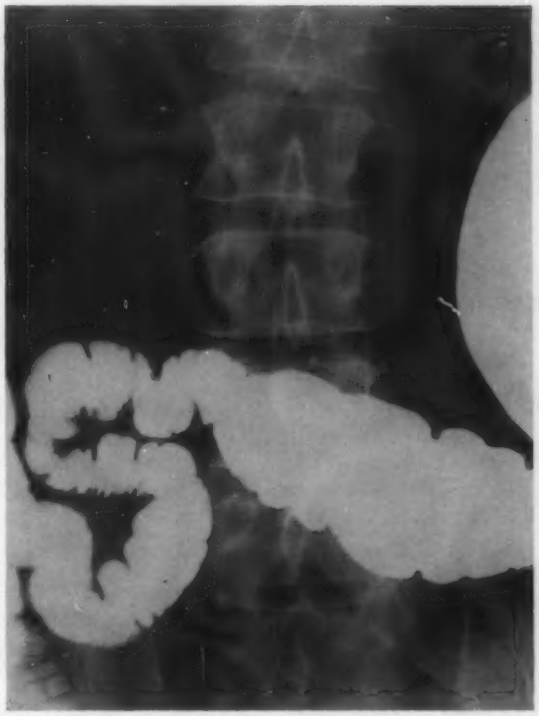

Fig. 5. Case III. One year earlier this patient had a right hemicolectomy for carcinoma of the cecum, followed by wound dehiscence with infection and a residual chronic draining sinus. A subsequent barium enema roentgenogram shows a segmented bony structure in the epigastrium measuring 7.0×1.0 cm., at the right lateral aspect of L2 and L3. It appears to arise from the vertebral bodies and bridge the disk space. There are two segments forming a sickle-shaped structure and separated by a space resembling a "pseudoarthrosis." The proximal segment is "Y" shaped at its upper end (not well seen in this view).

Case VI. J.M.A., a sixty-seven year old white male, was examined in 1956, at which time he gave a thirty-five year history of chronic peptic ulcer. In 1922 an operation for "excision of a peptic ulcer" and an appendectomy had been performed. In 1932 a gastroenterostomy was done. He continued to have intermittent pain and episodes of gastrointestinal bleeding and had numerous hospitalizations. His complaints were readily controlled with medical therapy. There were no symptoms relative to the surgical scar in the right upper abdominal quadrant. A year later the patient died in another hospital and gastric carcinoma was found at postmortem examination. There was no examination of the surgical scar.

Roentgenograms of the abdomen (1956)

Fig. 6. Case III. Roentgenogram in a tangential view localizes the bone (seen at the left) in the anterior abdominal wall. The opaque material (seen at the right) was injected into an infra-umbilical sinus tract and has outlined an irregular abscess cavity which has no communication with the bone. The distal end of the bone is fenestrated. Its appearance in this case is characteristic of these bone formations as visualized in the tangential view.

showed a fragment of bone lying obliquely in the plane of the right transverse process of the second and third lumbar vertebrae. Pancreatic calcifications were also present in the same area. On an oral cholecystographic study the bone was visible in the plane of the neck of the opacified gallbladder (Fig. 12). Only the tangential view of the abdominal wall permitted its exact localization. However, this interpretation was made only in retrospect. Until then the bony shadow was thought to represent variously a fish or animal bone in the gastrointestinal tract, a calcified cystic duct, a biliary calculus, a bone fragment separated from the lumbar

Fig. 7. Case III. Roentgenogram in the oblique view shows the bone overlying the right renal shadow.

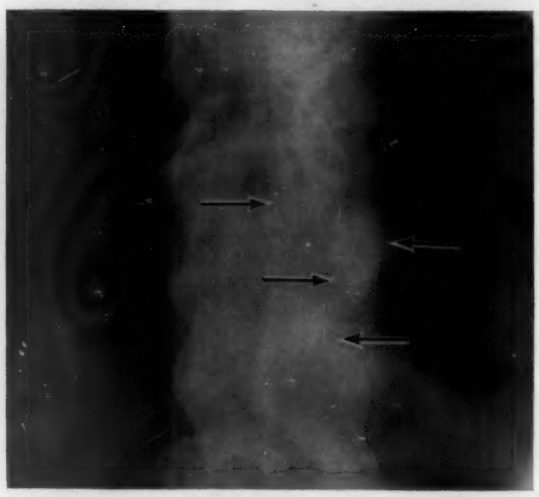

Fig. 8. Case IV. Superimposed on T11 and T12 is an 8.0×2.0 cm. bony structure "articulating" with the xiphoid at an angle of 135 degrees. It is poorly visible in this posteroanterior view and simulates a large irregular osteophyte associated with the vertebral bodies (arrows). The tangential view, which confirmed its location in the laparotomy scar, showed two small fenestrations. This was discovered nine weeks after subtotal gastric resection. (This roentgenogram is from a follow-up study made two years after detection.)

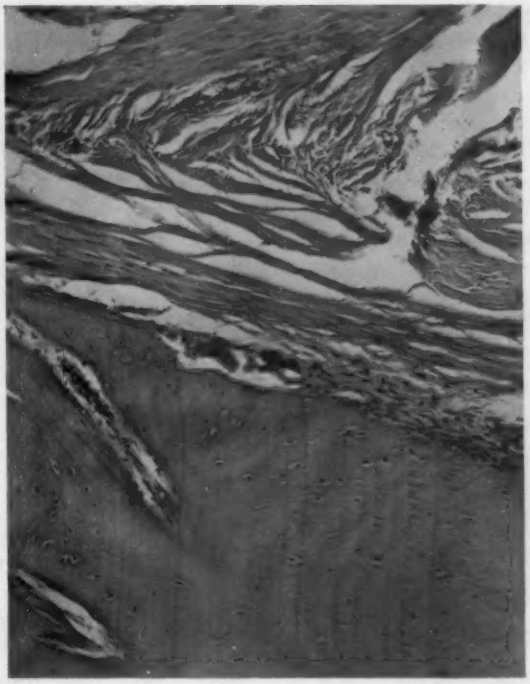

Fig. 9. Case v. Histologic section of a mass removed from an old right upper rectus scar reveals mature bone structure.

Fig. 10. Case v. Barium enema roentgenogram, made one year after repair of a perforated duodenal ulcer, shows a spindle-shaped process, suggestive of bone, measuring 8.4×1.5 cm. and overlying the left transverse processes of L1 and L2. This was the site of a hernia associated with the right upper rectus incision. The structure was distinctly visible on the posteroanterior roentgenogram, but a previous anteroposterior roentgenogram made for visualization of the lumbar spine did not show the bone clearly, since it was partly obscured by the vertebral bodies and the mottled soft tissue shadows cast by gas in the stomach and transverse colon.

Fig. 11. Case v. Oral cholecystogram in the oblique view shows the same structure in the plane of the common bile duct. The gallbladder and the heterotopic bone are not appreciably different in roentgenographic density.

spine or a calcified process associated with the right kidney.

Case vii. P.D., a forty-one year old white male with a history of chronic duodenal ulcer of three years' duration, developed an acute perforation and underwent repair on April 7, 1956. The operation was performed through a right upper paramedian incision. A small subcutaneous drain was left in place. The postoperative course was uneventful and the patient was discharged, improved, on April 18, 1956. He was re-admitted two years later (March 13, 1958) because of the recurrence of severe ulcer symptoms and tarry stools. A gastrointestinal examination revealed a deformed duodenal bulb. An oral cholecystogram showed a well-functioning gallbladder without stones.

Fig. 12. Case VI. A 3.0×0.3 cm. calcific shadow with distinct "cortical" margins overlies the right transverse process of L2. Several small calcifications nearby are in the pancreas. Note the relationship of the bone to the opacified gallbladder. An operation for peptic ulcer had been performed thirty-four years earlier and a gastroenterostomy had been done twenty-four years earlier.

Fig. 13. Case VII. Oral cholecystogram two years after the repair of a perforated duodenal ulcer shows a curvilinear quill-shaped shadow overlying the right transverse processes of L2 and L3. This bears a striking resemblance to the common bile duct.

Fig. 14. Case VII. In the oblique view of the same examination the position of the shadow changes from the medial to the lateral aspect of the opacified gallbladder.

On the posteroanterior study of the gallbladder there was a curvilinear, quill-shaped structure measuring 6.0×0.5 cm. This was of the same roentgenographic density as the gallbladder and was situated medially to it in the manner of an opacified common duct. However, on the oblique roentgenograms it assumed a position lateral to the gallbladder and was therefore recognized as a fragment of heterotopic bone in the right paramedian laparotomy scar (Fig. 13 and 14). The patient had offered no complaints referable to the scar itself. A subtotal gastric resection was done and the bone was excised with the scar.

Case VIII. W.G., a sixty-eight year old white male, was admitted on March 1, 1957, for recurrent epigastric distress and tarry stools. Four years earlier a gastrectomy had been performed for peptic ulcer. Previous operations included bilateral inguinal herniorrhaphy, prostatectomy and appendectomy. Examination of the abdomen revealed a nontender left upper paramedian scar, extending from the xiphoid to the umbilicus. There were also right lower quadrant

Fig. 15. Case VIII. Barium contrast examination of the upper gastrointestinal tract four years after subtotal gastrectomy shows a large mass of osseous character enmeshed in a row of metallic sutures. This tapers bluntly and shows "segmentations" demarcated by small, thorn-like projections arising in pairs from the sides of the bone. A distinct trabecular pattern is disclosed.

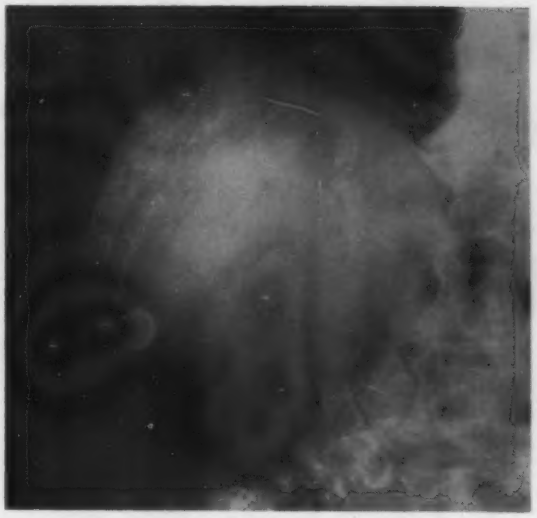

Fig. 16. Case IX. Oral cholecystogram four years after subtotal gastrectomy reveals nonvisualization of the gallbladder. A segmented structure, recognizable as bone, is seen to the right of L1 and L2. This measures 8.5×1.5×1.0 cm. and consists of a smaller proximal fragment and a larger distal fragment separated from each other by a small "pseudoarthrosis." A lamellated calculus measuring 1.5 cm. is seen 8 cm. to the right of the heterotopic bone.

and bilateral inguinal scars. None of the scars were tender and no masses or abnormalities in the left upper paramedian scar were found. The patient offered no complaints referable to the latter. He was treated medically and discharged.

During his hospital stay an examination of the upper gastrointestinal tract on March 5, 1957, disclosed a large bony mass to the left of the upper lumbar spine (Fig. 15). This was correctly interpreted as a scar ossification.

Case IX. E.A., a seventy-one year old white male, was admitted on December 30, 1957, because of generalized pruritus and right upper quadrant abdominal pain. Four years previously a subtotal gastrectomy had been performed for relief of persistent pain from a duodenal ulcer. There were no symptoms referable to the gastrectomy scar.

Roentgen studies of the gallbladder showed nonvisualization. The roentgenograms of the right upper quadrant showed a large calculus and a bony mass (Fig. 16 and 17). The latter was readily identified as being within the laparotomy scar. Cholecystectomy and choledocho-

Fig. 17. Case ix. In the oblique view the bone and the calculus are in the same plane.

duodenostomy were performed and gallstones were removed. The bony mass was not excised.

Case x. A.M., a fifty-five year old white male, had a laparotomy on January 15, 1958, through a midline incision extending from the ensiform cartilage to the umbilicus. Jejunitis and lymphadenitis of the jejunal mesentery were found. The postoperative course was uneventful. On June 30, 1958, the patient was again hospitalized for recurrence of symptoms of diarrhea, vomiting and weight loss. At this time examination of the laparotomy scar revealed a hard, nodular, superficial mass at its cephalic end. This mass measured approximately 5×3 cm., was freely movable and was regarded as a "calcifying hematoma of the operative wound" by the examining physicians who requested a roentgen examination of the area.

A tangential roentgenogram revealed bone in the scar but this could not be identified on the frontal projections because of direct superimposition on the lumbar spine which was extensively involved by Paget's disease. Review of earlier roentgenograms of the upper gastrointestinal tract showed that the bone could have been identified in the lateral view of the stomach and on roentgenograms of the esophagus (Fig. 18). Bony architecture was recognizable and the fragment measured 5.5×1.5 cm. Its lower end was forked. Subsequent gallbladder studies also showed the bone, but it was not superimposed on the biliary structures because of its relatively high position in the upper segment of the scar. The patient was asymptomatic with respect to the scar and removal was not considered necessary by the surgeons who saw him in consultation because of the mass. The patient's intestinal symptoms worsened and he expired on November 20, 1958. At the time of death the clinical diagnosis was malabsorption syndrome of undetermined origin. The mass in the epigastrium was removed at postmortem examination. Histologic section revealed mature bone.

In this patient the bone is known to have formed within a period of six months following operation.

THEORIES OF ORIGIN

Chauvin and Rouslacroix[10] have observed that theories explaining the formation of heterotopic bone in abdominal scars are almost as numerous as the case reports. However, in essence, all of the theories fall into either of two general groups. One theory is that the new bone arises from periosteum which has become detached from a neighboring bone; the second that

Fig. 18. Case x. Left oblique roentgenogram of the esophagus shows the bone superimposed on the lower right lung field (arrows). This bone was not visible on frontal abdominal roentgenograms because of superimposition on the vertebral bodies.

connective tissue cells, under certain conditions, may undergo metaplasia and become bone forming cells.

According to the first theory, periosteum may be detached from the xiphoid or pubis during surgical operation or as a result of muscular pull upon the ribs. There are, however, instances in which bone has formed in abdominal incisions where this theory is not applicable because the incisions have not involved the bone of either the symphysis or the pubis. To explain the latter cases, proponents of the theory of periosteal derivation point out that the phylogenic representations of the forward extension of the ribs are the tendinous inscriptions and similarly that the downward extension of the sternum is represented by the linea alba. They maintain that both the tendinous inscriptions and the linea alba may occasionally contain osteogenic elements, which, lying dormant, are stimulated to activity and proliferation to bone by the hemorrhage, trauma and inflammation which accompany the surgical incision. Furthermore, they postulate that when calcium and phosphorus salts, an increased blood supply and other appropriate biochemical conditions are present, bone formation is likely to take place.

The metaplastic theory, on the other hand, strongly championed by Leriche and Policard[24,25] and also supported by Weinmann and Sicher,[31] assumes that probably all loose connective tissue contains reserve cells, the undifferentiated mesenchymal cells of Maximow, the pluripotentiality of which is well known. Reactive proliferation of young connective tissue or granulation tissue is undoubtedly present in all healing incisions. In proliferating loose connective tissue where reserve cells undergo rapid mitotic division, a great number of pluripotential connective tissue cells are present. Under the influence of different stimuli, these cells can be induced to differentiate into macrophages, fibroblasts, chondroblasts, or osteoblasts, or remain in their state as reserve cells. Whether the process is one of indirect metaplasia (fibroblasts to cartilage to bone), or of direct metaplasia (fibroblasts to bone), or of both, is probably of little clinical significance. Most authors feel that the process of formation of heterotopic bone is probably the same for all the sites of location in the anterior abdominal wall.

As a corollary to the fibroblastic metaplasia theory, Keith[20] postulated that the cells which assume a bone-forming role are derived from capillary system endothelium or reticuloendothelium. He considered the first requisite for bone formation to be the presence of a condition which would stimulate the neighboring capillary system to throw out processes. In the invading vascular buds, he felt there were cells which could become both osteoblastic and osteoclastic in their action. This view is not as widely held as that of metaplasia of the pluripotential connective tissue cells.

Inflammatory reaction,[1] foreign bodies such as catgut,[14] wound infection, incisional hematoma and accidental implantation of urinary bladder mucosa[16,23] have all been implicated as significant factors in heterotopic bone formation. However, the frequency of such bone formation is much less than one would expect if any of these conditions were actually essential precursors of the process. Implanted acid gastric secretion, also proposed in the past as a mechanism for bone formation, was shown by Gruca[17] to be unimportant when he demonstrated that repeated injections of acid pepsin solutions into abdominal wounds of experimental animals produced no bone within the wound. Moreover, cases of bone formation have followed cholecystectomy where acid gastric secretion is not expected to be a factor. It is well known that transplanted bladder mucosa may result in ossification.[16,23] Other factors which have been considered important in the formation of heterotopic bone are an "inherent tendency" to new bone formation, metabolic imbalance, glandular disorders, parathyroid gland dysfunction and changes in the acid-base balance of the blood.[29] None of these mechanisms can be upheld in any

significant number of cases, however.

Extraskeletal new bone formation has also been found in the eye, lung, heart muscle, blood vessels, urinary bladder, kidney, nerve tissue, fascia, tendon, skeletal muscle, meninges, arachnoid, tonsil, thyroid, ovary, uterus, fallopian tube, penis, intestinal wall, appendix and in the dental pulp.[20,30] However, in these structures the development of the bone is not usually preceded by surgical treatment.

SYMPTOMATOLOGY AND DIAGNOSIS

It is agreed by most observers that heterotopic bone formation in surgical abdominal wounds is usually asymptomatic. The patient may, however, complain of some tenderness in the wound. Most characteristically, pain is present in the area of the scar when the patient bends forward, but is absent in the erect position.[3,5,6,7,11,15,18,30] Pain on position change is apparently related to the pressure of the bone on neighboring structures such as the xiphoid, rib cage, or soft tissue.

On physical examination a hard, tender or nontender mass may be palpated. This must be differentiated from the hard mass due to recurrent neoplasm in the incision. Occasional instances of bone formation coexisting with recurrent tumor tissue in the surgical scar have been observed.[13,14]

The frequency of abdominal scar ossification has been investigated by Mebius,[26] who studied 31 laparotomy scars macroscopically and microsopically at postmortem examination and found varying degrees of bone formation in three of the scars. The frequent lack of local symptoms probably accounts for the small percentage of cases discovered, despite the great number of abdominal operations which are performed. Sanders[29] described his first case as a "unique experience," because he had not seen such bone formation during thirty years of surgical practice. He then went on to discover 6 cases in the ensuing seven years. Actively aware of the condition, Chiariotti[11] found 6 cases in a series of 64 laparotomies, an incidence of 9.4 per cent. Chauvin and Rouslacroix[10] state that after becoming aware of the condition they observed 5 examples in three years without systematically searching for this lesion. Thus it is likely that these bone formations occur much more frequently than the literature indicates and are often missed by the clinician, surgeon, roentgenologist and pathologist because of lack of awareness of the existence of this entity.

PATHOLOGIC FINDINGS

Numerous histologic studies of the excised specimens have invariably revealed true bone, usually of the cancellous type.[1,15,19,30] Roentgen studies of excised specimens also show typical cancellous bone. Where roentgenograms of the living patient are of good quality, characteristic trabecular bony structure may be readily identified. The bone has been found at different depths within the anterior abdominal wall, *i.e.*, superficially located within the scar in or near the skin, at the level of the fascial covering of the muscles or within the muscle tissue proper. Initially, the bone is produced in multiple minute foci which coalesce and form irregular bony plates of varied size.[9,10] In his review of 30 cases, Bouton[9] found occasional involvement of the entire scar. In other instances the osseous tissue was very small and at times even microscopic in size. His largest specimen measured 10 cm. in length. There is also considerable variability in the shape and contour of the formations, which may be plaque-like, round, elongated, regular or irregular. Both Goldstein[15] and Silver[30] noted "Y" shaped or prong-shaped formations. Most cases occur above the umbilicus.[1,6,19] Some have formed in the abdominal incision as early as three weeks following surgery, while in other instances as much as fourteen years have elapsed before detection.[8,21,27] Most cases are probably not discovered for years and hence there is a tendency to regard them as having formed shortly before detection. However, it is more likely that the bone forms within weeks or only a few months after

operation in practically all cases.[11] Silver[30] feels that the osseous tissue may remain permanently in most instances but that some formations may eventually be completely resorbed. He therefore recommends a waiting period before surgical excision in order to allow sufficient time for possible resorption, provided removal of the mass is not indicated because of troublesome symptoms. Bouton,[9] on the other hand, states that although bone formations in muscles of other parts of the body have been known to decrease in size and eventually disappear spontaneously, as in post-traumatic myositis ossificans, this has not been observed in ossification of abdominal scars. Recurrence following surgical excision has not been recorded.

TREATMENT

Surgical removal of the bony mass, if symptomatic, is the only known form of treatment. There appears to be no contraindication to allowing the bone to remain.

ROENTGEN FINDINGS AND DIFFERENTIAL DIAGNOSIS

In 8 of the 10 cases the roentgen images of the scar ossifications in the roentgenograms of the abdomen, gastrointestinal tract or gallbladder were readily recognizable as bone since they showed typical cortical and medullary structure. In the 2 exceptions the osseous tissue, lacking the distinctive appearance of bone, or even of a calcific process, was indistinguishable from the density of the opacified gallbladder (Fig. 2 and 11). This could conceivably be a source of error, particularly if plain roentgenograms of the abdomen or right upper quadrant were not made prior to examination of the gallbladder. In one of these 2 cases (Case VII) the common bile duct was simulated perfectly.

In several cases the posteroanterior or anteroposterior roentgenograms of the abdomen did not show the heterotopic bone at all (Case 1 and X) or obscured it considerably because it was superimposed upon the vertebral bodies (Case II and V). However, it was visible on subsequent oblique roentgenograms and became a potential source of confusion in the interpretation of biliary tract examinations.

Thus, it becomes important to bear in mind that patients who have had surgical procedures performed through upper abdominal incisions may develop ossification in their scars and that these may be noted for the first time superimposed upon right upper abdominal structures since they are not immediately recognized or are actually invisible in frontal projections of the abdomen. In those cases where the heterotopic bone is indistinguishable from the common bile duct because of its lack of visible bony trabecular pattern, it may be differentiated from the duct only on roentgenograms made in the extreme oblique position where it assumes a position to the right of the opacified gallbladder. Finally, the tangential view of the anterior abdominal wall will confirm the presence of heterotopic bone in the scar.

It is possible that a mass palpable in an upper abdominal scar may represent bony matrix which is insufficiently calcified to be visible on roentgenograms made in the early postoperative period.[3,9] Rauhs[28] states that even as early as three weeks after an operation an area of increased density at the site of the scar becomes visible in the roentgenogram and that this later becomes recognizable as bone. We have not seen any examples of this type.

The differential diagnosis from other calcified structures in the upper abdomen is relatively simple. Osteophytes originating from the lower dorsal or upper lumbar vertebrae may be distinguishable only in tangential or oblique views.

In those instances where readily recognizable bony tissue is visible in the spinal area, it is likely to be regarded as having originated in the vertebral column (Fig. 4, 5, 8 and 10).

When these bony masses are superimposed upon the vertebral bodies, but are still perceptible in the roentgenogram, they

must be differentiated from malformations, neoplasms and residuals of trauma or infection of the spinal column (Fig. 4). It is particularly important to keep this entity in mind when dealing with the numerous spinal studies which are routinely made for complaints of back pain, especially following injury.

In a patient seen recently, a mass of heterotopic bone, simulating closely the type found in the laparotomy scars, was noted. This proved to be within the superficial soft tissues of the paraspinal area, resulting from an old stab wound of the back.

Extensively calcified costal cartilages usually lie in the oblique position pointing downwards and outwards, away from the midline, whereas the ossifications within scars are usually vertical in position. Calcified costal cartilages also tend to be multiple or paired, with bilateral, symmetric patterns. The calcified material is usually granular in character, unlike the scar ossifications. However, trabecular bony structure is often present in costal cartilage and in such instances this may be indistinguishable from scar ossification in the frontal or oblique views.

Limy bile in the common duct may very rarely be revealed as a shadow in the right upper quadrant and thus may simulate a piece of heterotopic bone.[12] In our own experience with one such case, the shadow was of equivalent density and structure to that noted in Case VII (Fig. 13 and 14), inasmuch as it lacked true bony texture.

Additional features which may aid in the recognition of heterotopic bone formations are the gross structural details as exemplified in our cases. These include the presence of fenestrations, segmentation with formation of a joint-like relationship between the segments ("pseudoarthrosis") thorn-like, branching projections from the main body, a curved, spindle or fusiform configuration and a tendency toward tapering of the mass at each end or a "Y" configuration at one end.

SUMMARY

Ten cases of bone formation in upper abdominal laparotomy scars have been presented and theories regarding the formation of such tissue have been reviewed and discussed.

None of the patients offered complaints referable to the bone in the scar and elective removal was not done in any case. This is contrary to previous reports in which there are frequent references to the occurrence of pain on forward bending.

Lack of recognition of the presence of the bone led to roentgenologic and clinical misinterpretation of findings. In this series of 10 cases there were 5 instances (Case I, II, III, IV, and V) in which the heterotopic bone, although demonstrable in the roentgenograms, was not identified as scar ossification. The period of delay in recognition ranged from one month to twenty-five years. In 2 patients (Case VIII and IX) there were elapsed intervals of four years prior to recognition of the ossification in the scar. In these, it could not be determined whether other roentgen studies which may have revealed the bone had been done during the interval between surgical treatment and roentgenologic recognition of the heterotopic bone. In the remaining 3 cases the heterotopic bone was recognized as such on the first roentgen studies made postoperatively.

This entity should be differentiated from osteophytes, malformations and traumatic bone fragments arising from the lumbar spine, from scars of the back, from calcified costal cartilage, foreign bodies in the gastrointestinal tract and, particularly, from the opacified common bile duct.

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ADDENDUM

Since this paper was submitted for publication an additional instance of bone formation in a left upper rectus abdominal scar has been found.

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FAMILIAL RECURRING POLYSEROSITIS*

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FAMILIAL recurring polyserositis is a disease whose nomenclature has undergone tortuous deviations, a circumstance common to the emergence of a new entity of which the etiology and pathogenesis are unknown.

From the original reference to the disease in 1908 as "paroxysmal syndrome,"[7] the various appellations have ranged through "benign paroxysmal peritonitis,"[15,16] "periodic abdominalgia,"[12] "periodic peritonitis,"[13] "la maladie périodique,"[1,4,5,8,9,10] "la maladie dite périodique,"[17,18] "epanelepsie Méditerranéenne,"[10] "familial Mediterranean fever,"[6] disease of Siegal-Cattan-Mamou,"[2] and "Armenian disease."[14]

Until such time as the etiologic-pathogenetic features of the disease are elucidated, we prefer the designation familial recurring polyserositis[11] inasmuch as it best describes the cardinal clinical aspects of a condition which is familial, of a recurring nature and presents acute inflammation of not only the peritoneum but multiple serous membranes.

CLINICAL FEATURES

Familial recurring polyserositis is a heredofamilial disorder limited almost exclusively to Armenians, Arabs and Jews. The onset of the disease is for the most part before the third decade although a minority of patients present initial symptoms in the fourth and fifth decades. The disease is found about twice as commonly in males. Acutely painful attacks involving the abdomen, chest or joints, and occurring in an episodic, repetitive fashion, characterize the disease. In the interim between attacks, which vary greatly in frequency, the patient is usually entirely well.

The most common manifestation of this disease is the acute abdominal attack. Although often presaged and accompanied by malaise, anorexia, nausea, or feeling of abdominal distention, the keynote symptom is pain, which may be felt in any quadrant of the abdomen. It may remain localized or spread diffusely over the abdomen in the course of an attack which rarely exceeds two to three days' duration. The pain is steady but may have periodic accentuations, particularly with body movements which the patient attempts to avoid. The pain is of such severity as to require opiates for any degree of amelioration.

Fever, although occasionally absent, most often is present to as high as 104°F. Decreased bowel action is the general rule, but on occasion mild diarrhea may occur, more often in the terminal phase of the attack.

Physical findings in addition to the usual fever include abdominal signs of local or diffuse direct and rebound tenderness, involuntary muscle guarding, decreased to absent bowel sounds and varying degrees of abdominal distention. Clinical icterus is occasionally noted and hepatosplenomegaly of slight degree can most often be delineated. Residual splenomegaly of slight to moderate proportions occurs in some cases of long standing disease.

As an adjunct to the abdominalgia, or occurring at times as a separate event, is the second most common manifestation of familial recurring polyserositis; namely, pleuritic chest pain. The pleuritis responsible for this pain may be associated with a small pleural effusion. Physical findings for the most part are limited to suppression of breath sounds over the basal area of the

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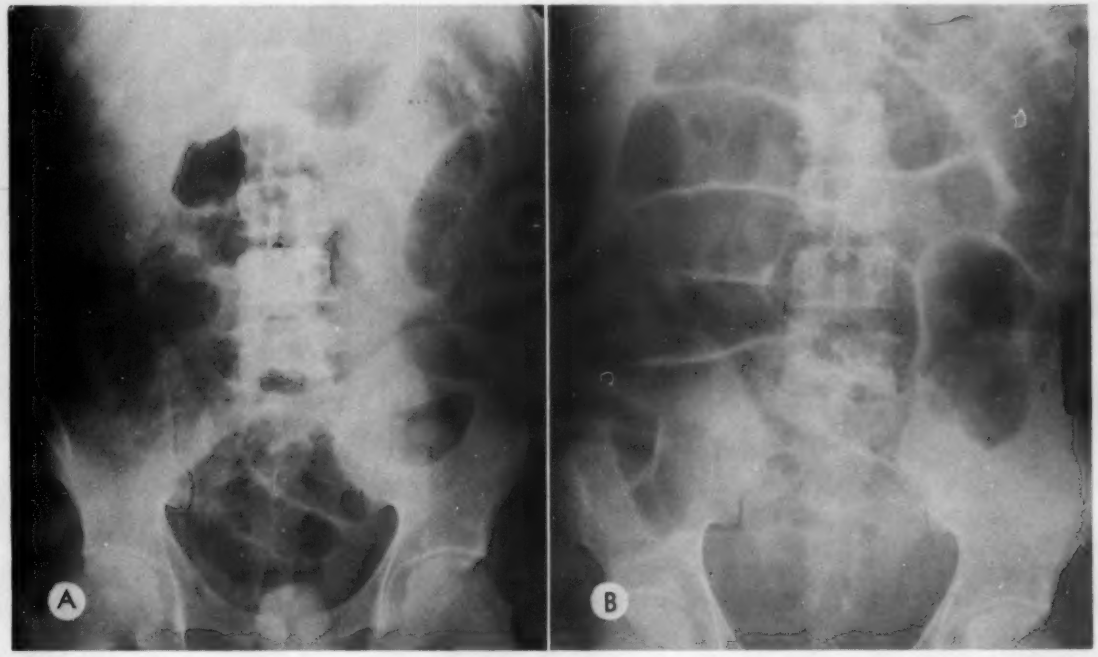

Fig. 1. Intestinal ileus during an attack of acute abdominal pain. (*A*) Supine roentgenogram made during the first few hours of the attack of pain demonstrates air distention of the small intestine. (*B*) Supine roentgenogram four days later shows marked increase in small bowel distention, and practically no gas in the colon. The appearance is consistent with mechanical or paralytic ileus involving the small intestine.

involved side due to splinting of the diaphragm.

Joint symptoms are present in a variable number of patients with familial recurring polyserositis and range from vague arthralgias to an acute arthritis. One or more major joints may be affected and the pattern of the attack is similar in abruptness of onset, duration and interval well being to the previously described abdominal and thoracic manifestations.

Other serous membranes such as pericardium and meninges may be rarely involved in the recurrent, brief, acute inflammatory episodes which characterize the disease.

The known pathology of familial recurring polyserositis, derived mainly from misguided abdominal surgery, consists of a nonspecific acute serofibrinous visceral and parietal peritonitis. It is assumed that other serosal surfaces present a similar nonspecific acute inflammatory reaction. A secondary type of amyloidosis with resulting renal death has been reported as a terminal complication in a few patients.

Ancillary laboratory data during the acute attacks include a leukocytosis with neutrophilia, increased sedimentation rate and increased blood fibrinogen. We have found a positive Harrison spot test for bilirubinuria in the majority of attacks. Chromatographic studies of the urine are being pursued in an attempt to find a specific abnormality.

In the absence of any specific laboratory test, the clinical diagnosis of familial recurring polyserositis is based upon the classic features of the attack which is of severe intensity, brief duration, with rapid and complete resolution, stereotyped recurrence and absence of detectable etiology.

Roentgenologic Manifestations

Roentgen examinations are available for study in 17 of the 20 cases which have been seen at our hospital. The positive examinations which have been obtained are those

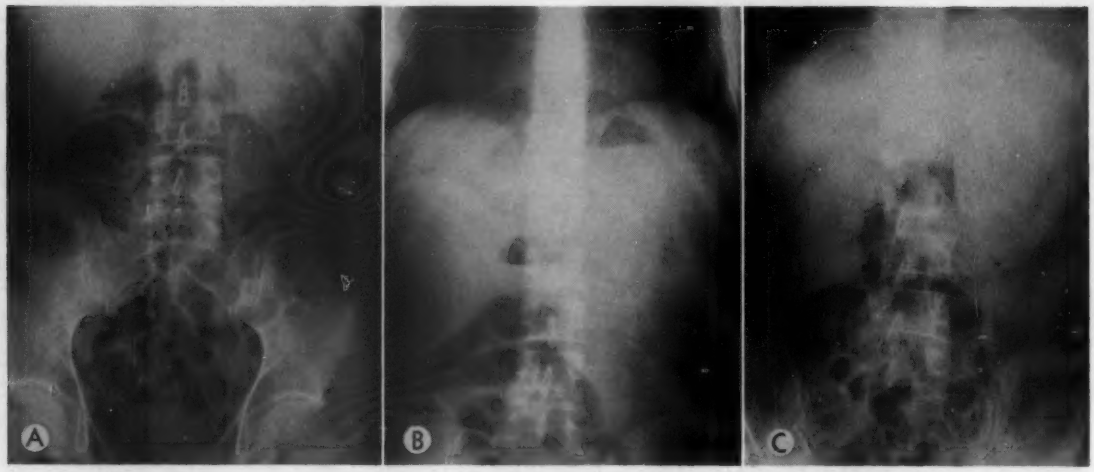

FIG. 2. Small intestinal ileus during an attack of acute abdominal pain. (*A*) Supine roentgenogram shows air filling of the small intestine with moderate distention of the upper small intestine. There is some gas in the colon. (*B*) Upright roentgenogram demonstrates the small intestinal ileus and shows air fluid levels. (*C*) Left lateral decubitus roentgenogram provides a somewhat better demonstration of air fluid levels in small intestinal ileus.

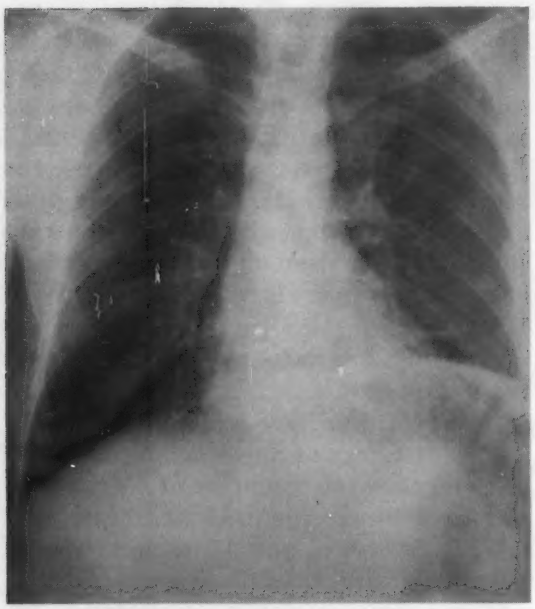

FIG. 3. Examination of the chest during an acute attack of left chest and abdominal pain. A postero-anterior roentgenogram of the chest shows atelectasis at the left base with a minimal amount of fluid in the left costophrenic angle. The left leaf of the diaphragm is elevated and beneath it can be seen a dilated loop of air filled small intestine. These findings clear completely during the few days following the attack of pain.

of the abdomen, chest and the small intestine. Only one of our patients had clinical evidence of joint involvement, but joint studies have not been obtained. The area most commonly involved is the abdomen and this region is thus the one most often examined. Nine patients roentgenographed during acute abdominal attacks have had air distended loops of small bowel. Figure 1*A* shows a fairly typical appearance with small amounts of air present in the large intestine, but with air distention of several loops of small bowel. This patient's attack increased in severity and Figure 1*B* shows the appearance four days later at which time the colon is practically devoid of air and there is marked distention of many loops of small bowel with a pattern consistent with mechanical small bowel obstruction.

Figure 2 shows (*A*) supine, (*B*) upright and (*C*) left lateral decubitus roentgenograms of an abdominal study during an acute attack of pain in which there are air fluid levels in the small bowel and an appearance consistent with that of a partial mechanical small bowel obstruction or of a paralytic ileus. Clinically the liver and spleen are thought to become more promi-

nent during the attack, but no roentgenographic evidence of this change in size is available. In a number of the patients, however, the spleen appeared enlarged. The distention of the small bowel has been noted previously by Brick and Cajigas[3] and by Heller *et al.*[6]

Five of our patients have shown positive findings in roentgenograms of the chest made during attacks of chest pain. These attacks have usually been concurrent with an attack of abdominal pain. Figure 3 shows basilar atelectasis accompanied by minimal fluid, elevation of the left leaf of the diaphragm and small intestinal distention. Figure 4 shows a moderate right pleural effusion and accompanying atelectasis. This is the largest pleural effusion in our group of cases. As would be expected, fluoroscopy demonstrated limitation of diaphragmatic motion on the affected side. Siegal[16] has reported fluid in the pleural space previously.

In an attempt to learn more about the pathologic physiology of these patients, 13 of them have had barium studies of the small intestine during attacks of abdominal pain. The demonstration of motor abnormalities in the small bowel shown by barium study has been very briefly mentioned by Reimann[12] and Bickel and Lasserre.[2] In a number of instances, patients were unwilling or unable to ingest the full amount of barium. A considerable delay in over-all transit time of barium to the colon was noted in most of these studies. When this has occurred, there has in general been a discontinuous column and dilatation of many loops with more residual barium snowflakes left behind than usual. Some of these loops also gave the appearance of some thickening of mucosa and the discontinuous column was on occasion connected by strings of barium.

Although the column is interrupted, the appearance is distinctly different from that of the flocculation of "deficiency" disease. The small intestinal loops lose their normal regular neighborly arrangement and the large dilated loops extend across either side

Fig. 4. Examination of the chest during an acute attack of right chest and abdominal pain. There is atelectasis at the bases and a moderate right pleural effusion; this is the largest fluid collection in our series.

of the abdomen. The greater the dilatation and the discontinuity, the more bizarre and less orderly is the arrangement of the loops. Figure 5 illustrates (*A*) two and one-half hour, (*B*) five hour, and (*C*) seven hour roentgenograms of a patient in an attack of abdominalgia. The two and one-half hour roentgenogram shows slow progress of the barium, considerable disparity in size of the loops, discontinuity of the column and a coarse mucosal pattern. At five hours, the dilatation of the jejunum persists and there is more marked discontinuity. The barium has reached the cecum, however. At seven hours the jejunal distention and the other abnormalities are all still present. Transit is slow. This is a typical example of the more marked abnormalities. Figure 6 is a one-half hour roentgenogram of the same patient made during the interval between attacks. Note that there is no dilatation or disorganization of the barium pattern and that transit time is normal. This normal appearance between the attacks of pain has been found in all cases so examined. Figure 7 shows similar but less severe changes. (*A*) A three hour roentgenogram during an attack of abdominalgia shows

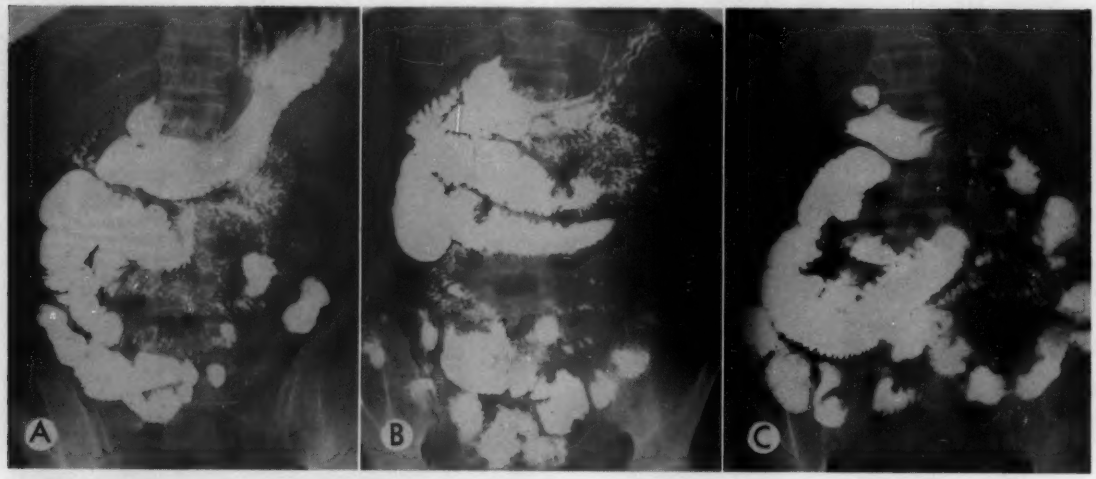

FIG. 5. Barium study of the small intestine during an attack of acute abdominal pain. (*A*) A roentgenogram made two and one-half hours after ingestion of the barium shows discontinuity of the column, dilatation of several loops and very slow transit of the barium through the small intestine. (*B*) Five hour roentgenogram again shows the considerable disparity in size of the loops and loss of the orderly arrangement. Some barium, however, has reached the cecum. (*C*) Seven hour roentgenogram shows persistent dilatation of the jejunum, discontinuity of the column and disparity in size of the loops with loss of the usual orderly arrangement.

FIG. 6. Same patient as in Figure 5. Study of the small intestine during an asymptomatic interval. This roengenogram was made one-half hour after the ingestion of the barium. The small bowel is within normal limits in every respect. This is typical of the appearance of the small bowel during the intervals between attacks of pain.

only the jejunum filled, and demonstrates many widened loops. (*B*) The one-half hour roentgenogram during the asymptomatic interval is within normal limits. Figure 8 (*A*, *B* and *C*) shows minimal changes during an attack of pain. There is disparity in size of the loops but only moderate discontinuity.

Because it was felt that the presence of pain by itself might be responsible for the findings in the barium studies of the small intestine, an attempt was made to find small bowel studies done on patients with severe abdominal pain of other origin. Only a small number of such cases could be found, but in every instance, the small bowel pattern was within normal limits.

SUMMARY

1. In a patient of Armenian, Syrian or Jewish background who presents with an attack of pain which could be caused by inflammation of one of the serosal surfaces and, in particular, if he gives a history of recurrent attacks, familial recurring polyserositis should be considered. The radiologist can be of some assistance in making this diagnosis.

2. Roentgenologic findings in familial

FIG. 7. Comparison of the appearance of the small intestine during abdominalgia and during a pain free interval. (*A*) This roentgenogram was made three hours after the ingestion of barium during an attack of pain. It shows changes similar to those seen in Figure 6, but somewhat less marked. The transit of barium is slow and the jejunum is dilated. The mucosa appears thickened and the arrangement of loops is not orderly. (*B*) This roentgenogram was made during an asymptomatic interval and shows the loops to be of normal caliber and mucosal pattern and shows small amounts of barium having reached the colon at one-half hour. This is considered to be entirely within normal limits.

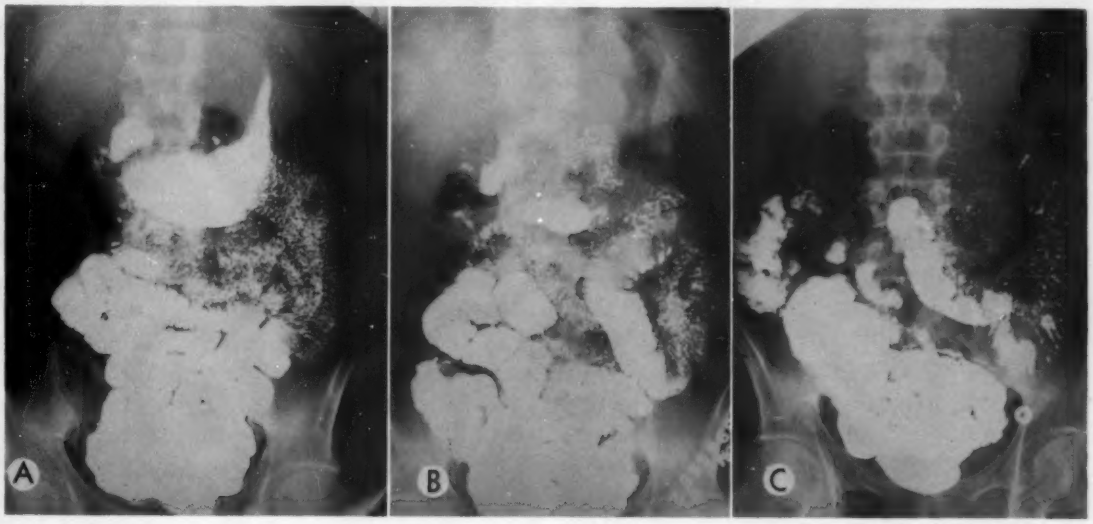

FIG. 8. Barium study of the small intestine during an attack of pain. The changes in the small bowel are considerably less marked in this examination than they are in the other studies illustrated. There is, however, disparity in size of the loops and discontinuity of the column. (*A*) Roentgenogram made at two and one-half hours. (*B*) Roentgenogram made at three and one-half hours. (*C*) Roentgenogram made at five hours after ingestion of the barium meal.

recurring polyserositis are for the most part nonspecific, but are helpful in confirming a diagnosis. During attacks of abdominal pain, plain roentgenograms of the abdomen show small intestinal ileus and a barium meal study shows discontinuity of the column, dilatation and delay in transit. During attacks of pleural pain, basilar atelectasis is generally demonstrated and pleural fluid in small to moderate amounts is often found.

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ROENTGENOLOGIC FINDINGS IN FAMILIAL MEDITERRANEAN FEVER*

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RECENT publications have clearly delineated the clinical syndrome which we prefer to call familial Mediterranean fever. Patients afflicted with this disease seek medical attention because of attacks of acute pain in the chest, abdomen or joints. The aid of the radiologist is frequently enlisted for evaluation of the symptoms. Since roentgenologic findings have been erroneously interpreted as tuberculous pleuritis, intestinal obstruction, or tuberculous or rheumatoid arthritis, it is important that the roentgenologist be aware of the clinical as well as the roentgenologic manifestations of this entity if he is to play a part in establishing the diagnosis.

CLINICAL FEATURES

Familial Mediterranean fever is a familial disease affecting peoples of Mediterranean origin, mainly Non-Ashkenazi Jews and Armenians.[5,6]

The disease begins in childhood or early adolescence. It is characterized by recurrent attacks of fever accompanied by either abdominal pain, arthralgia, chest pain or skin manifestations. The attacks are of short duration, usually lasting from twelve to forty-eight hours, and recur at irregular intervals. Most cases suffer from all three forms of the attacks at various times. Between attacks the patients are well. The disease lasts for many years, and in a certain percentage of cases terminates in renal failure[8] and death due to amyloidosis.[6]

The clinical pictures, as well as the familial incidence, the limitation of the disease to certain ethnic groups, and the recessive transmission[7] seem to indicate that the basic disorder is an inborn error of metabolism.[7]

A rapid erythrocyte sedimentation rate, high fibrinogen levels[6] and slight spherocytosis[4] are the only laboratory findings.

Therapeutic measures are of no avail. No patient has been known to recover, but remissions of variable duration do occur.

The disease was described by Siegal[10] as "benign paroxysmal peritonitis." In the last decade cases reported from different Mediterranean countries were usually designated as "periodic disease."[1–3,9,11] A detailed account of the clinical picture, based on 74 cases seen at the Tel-Hashomer Hospital and 179 culled from the literature, was recently published by Heller, Sohar and Sherf.[6]

MATERIAL

Of 80 cases diagnosed as familial Mediterranean fever at the Tel-Hashomer Hospital, 40 have been examined roentgenologically because of one or more manifestations of the disease. The examinations were performed during attacks, during the abatement of attacks and during quiescent intervals. The material is divided into findings pertaining to attacks in the (1) abdomen, (2) chest, and (3) joints.

ROENTGEN FINDINGS

(A) *Abdominal Attack.* This form of attack begins with fever and abdominal pain which gains rapidly in intensity. Signs of peritoneal irritation appear which may progress to board-like rigidity. The patient is constipated and sometimes vomits. After twelve to thirty-six hours the patient is clinically well.

Abdominal roentgenograms were taken during the peak of the attack in 12 patients. In all of them, varying degrees of

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small and large intestinal distention with fluid levels were found. Control roentgenograms taken immediately after the attack subsided showed no deviation from the normal.

(B) *Chest Attack.* This may appear alone or concomitant with an abdominal attack. It consists of fever, pain of pleural character, shallow breathing, and dullness and decreased breath sounds over one of the lung bases. After twelve to twenty-four hours the attack subsides.

On roentgenologic examination pleural effusion on the side of the pain is seen without any evident changes in the lungs. The effusion is usually small and is rapidly reabsorbed over three to five days, leaving only signs of minimal pleural thickening.

CASE 1. Female, twenty-four years old. Roentgenogram taken during an attack in the left chest demonstrated left-sided pleural effusion (Fig. 1*A*) which cleared completely after five days (Fig. 1*B*). Ten days later, during an attack of pain involving the right side of the chest, pleural effusion was seen on that side (Fig. 1*C*).

(C) *Joint Attack.* Attacks of pain in the joints are more varied than those described above. One or more joints may be simultaneously involved. The affected joint is tender, immobile and may be red and swollen. The attack usually lasts for a few days and may be accompanied by swelling and effusion into the joint. It is not unusual, however, for one joint to remain immobile and painful for weeks or even months, while the temperature is subfebrile. In the vast majority of patients, even after frequent and prolonged attacks, complete clinical remission and return of mobility occur. Only rarely is there residual disability.

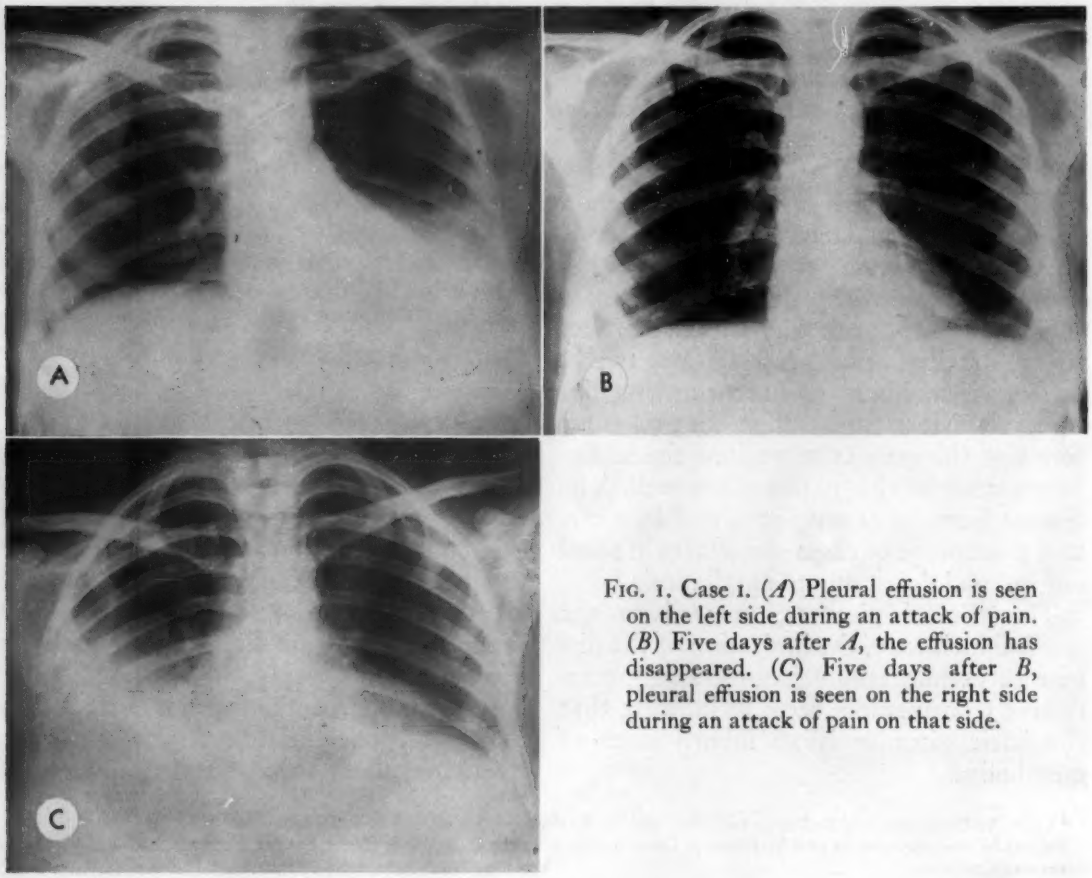

FIG. 1. Case 1. (*A*) Pleural effusion is seen on the left side during an attack of pain. (*B*) Five days after *A*, the effusion has disappeared. (*C*) Five days after *B*, pleural effusion is seen on the right side during an attack of pain on that side.

In short attacks there is usually no roentgenographic evidence of bone involvment; signs of soft tissue swelling and effusion in the synovial space may be observed. After more protracted attacks, subperiosteal osteoporosis is seen. Later, signs of destruction of the articular cartilage appear followed by narrowing of the joint space and osteophyte formation. The osteoporosis in the juxta-articular bones may progress to a marked degree and most probably is secondary to disuse of the affected limb during the acute phase. It is obvious that once roentgenologic signs of bone involvement do appear, they persist even when clinical remission ensues.

The sacroiliac joint, the hip, and the knee are involved with increasing frequency in that order. The shoulder, elbow, and ankle joints, although frequently the site of attacks, have never been found to show roentgenologic changes.

Case II. This fourteen year old male has suffered from familial Mediterranean fever since childhood. The major attacks of pain were located in the sacroiliac joints which showed signs of sclerosis and erosion of the adjacent ilium without narrowing of the joint space (Fig. 2). These roentgenologic changes have not progressed over a period of more than three years.

Fig. 2. Case II. Bilateral sclerosis of iliac bones adjacent to the sacroiliac joints, with multiple erosions.

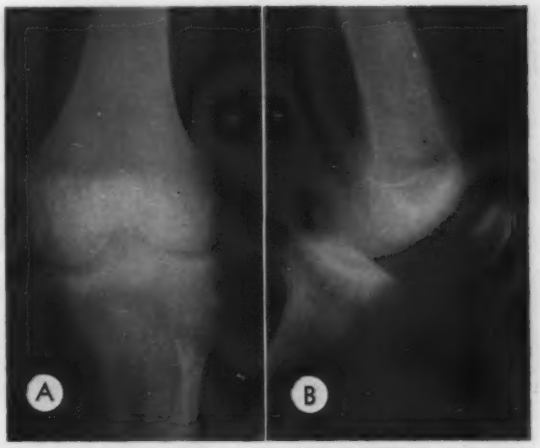

Fig. 3. Case III. (*A* and *B*) Subcortical and general osteoporosis of the right knee joint with narrowing of the joint space.

Case III. This twenty year old male suffered many attacks in his right knee. Roentgenographic examination showed some narrowing of the joint space, severe osteoporosis of the surrounding bone and osteophyte formation (Fig. 3). This led, in another hospital, to the diagnosis of tuberculosis of the joint and arthrodesis of the knee was performed. Pathologic and bacteriologic examinations revealed no evidence of tuberculosis; only a nonspecific reparative process was seen microscopically. The appearance of attacks of abdominal pain and other signs led ultimately to the diagnosis of familial Mediterranean fever.

Case IV. This thirty year old male has suffered from familial Mediterranean fever since childhood. After many attacks in his right shoulder over a period of several years, he developed a frozen shoulder. Severe osteoporosis of the humerus was demonstrated (Fig. 4*A*). The clinical symptoms remained severe for several months but after they subsided the patient was again able to move his shoulder with some limitation. The osteoporosis regressed, but not completely (Fig. 4*B*). The patient has also had attacks of pain in his right hip and right knee. The roentgenographic changes (Fig. 5 and 6, *A* and *B*) were similar to those previously described.

DISCUSSION

The roentgen findings observed during the abdominal attack are probably the expression of varying degrees of peritoneal

FIG. 4. Case IV. (*A*) General osteoporosis of the right humerus, with multiple erosions of the cortex from the inner side. (*B*) Six months after *A*, the porosis and erosions have diminished.

reaction which may progress even to transient paralytic ileus. They may be of diagnostic help when correlated with the clinical course.

The rapid appearance and disappearance of pleural effusion after a chest attack constitutes a highly significant roentgenologic finding. In a patient of Mediterranean stock, such a sequence of roentgen findings will permit the roentgenologist to suggest familial Mediterranean fever as a diagnostic possibility. The findings disappear quickly so that only daily examination for a period of several days after the attack will ensure a positive result.

The relative paucity of roentgen findings during the first attacks of arthralgia is due to the fact that only the joint cartilage and synovial membranes are involved. Roentgenologic changes have been detected only in the sacroiliac, hip and knee joints. In our series, no roentgenologic changes were seen in the shoulder, elbow and ankle joints, although they were frequently the site of attacks. The explanation may lie in the fact that the former are the major weight-bearing joints in the body. A parallel may be seen in the predilection of osteoarthritis for these joints. The degree of the osteoporosis is probably dependent upon the degree and duration of the limitation of movement of the joints.

In the differential diagnosis of the arthritic manifestations, the diseases of major importance are rheumatoid and tuberculous arthritis. In familial Mediterranean fever, in sharp contrast to rheumatoid arthritis, the small joints have never been

FIG. 5. Case IV. Roentgenogram of the right hip joint showing osteoporosis of the head and neck of the femur, as well as of the bony acetabulum, with narrowing of the joint space.

Fig. 6. Case iv. Roentgenograms of the right knee joint demonstrating (*A*) slight osteoporosis, especially subcortical, and (*B*) four years after *A*, marked osteoporosis, with narrowing and deformity of the joint space.

seen to be involved. Tuberculosis of the joint is difficult to differentiate on roentgenologic evidence alone. Knowledge of the clinical picture of familial Mediterranean fever is essential if an erroneous diagnosis of tuberculosis and unnecessary arthrodesis are to be avoided. A history of bouts of abdominal or chest pain is helpful, but this is sometimes lacking. In such a case, the diagnosis may depend upon an accurate family history, which alone will provide the clue.

SUMMARY

1. Roentgenologic evidence of attacks of familial Mediterranean fever may be found in the abdomen, the thorax and the joints.

2. Distention and fluid levels in the intestine have been seen during the acute abdominal attack.

3. Chest attacks are accompanied, as a rule, by pleural effusion. Its rapid disappearance strengthens the diagnosis of familial Mediterranean fever.

4. Awareness of the existence of familial Mediterranean fever is essential in the roentgenologic evaluation of joint attacks. Roentgenologic changes have been observed in the knee, hip and sacroiliac joints, and are similar in appearance to those of rheumatoid arthritis; however, in familial Mediterranean fever the small joints are not involved. Tuberculous arthritis may be differentiated by the discrepancy between the roentgen findings and the clinical manifestations.

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SARCOIDOSIS OF THE UPPER GASTRO-INTESTINAL TRACT*

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SARCOIDOSIS involving the upper gastrointestinal tract is a clinical and roentgenologic rarity. Recently, however, Palmer[8] has found pathologic proof of gastric sarcoidosis by mucosal biopsy in 10 per cent of 60 patients, none of whom had gastrointestinal complaints but all of whom eventually were diagnosed as sarcoidosis by the usual clinical and pathologic criteria. The biopsies were obtained by the vacuum tube method from fundic mucosa.

It would seem that if, as Palmer states, gastric sarcoidosis can be found in 10 per cent of sarcoid patients without upper gastrointestinal symptoms, then the number should increase among those who have upper gastrointestinal complaints. Positive pathologic and roentgenologic findings of gastrointestinal sarcoid have been rare. This could be due in part to the fact that these lesions often cause no gross changes; it also happens sometimes that roentgenographic changes are present but erroneously considered due to an unrelated entity in the mistaken belief that sarcoidosis does not affect the gastrointestinal tract.

A review of the pathologic, clinical and roentgenologic changes in gastric sarcoid and a presentation of a case therefore seems indicated in order to create a greater awareness that such pathology does occur, possibly more often than heretofore considered, and to arouse strong suspicion of this diagnosis in sarcoid patients with roentgenographic evidence of gastrointestinal pathology.

TERMINOLOGY—INCIDENCE—ETIOLOGY

Most of the nomenclature has been gradually shelved in favor of *sarcoidosis* or *Boeck's sarcoid. Benign lymphogranulomatosis* (or recognizable modifications of Besnier-Boeck-Schaumann's disease) is the preferred nomenclature in Europe.[4]

Although originally considered rare, sarcoidosis (other than gastric) is now known to be commonplace and has been reported in many countries throughout the world. It may occur at almost any age but is found most often in the age range from twenty to forty years. Cowdell[3] states that Schoenholzen (1947) had reported the presumptive rate in Swiss army recruits as 13 per 100,000.

The frequency of sarcoidosis in Negroes in this country has been found to be higher than would be expected on the basis of the population, 17.81 cases per 100,000 Negroes as compared to 0.88 per 100,000 whites in the United States Army.[7] In Europe, the Nordic groups exhibit the highest incidence.[4] Although not considered a familial disease, it has on a number of occasions been found in siblings.[4]

There is some difference of opinion as to what may accurately be listed as gastric sarcoidosis. Allen *et al.*[2] who have carefully reviewed the cases in the literature, feel that only 14 of the 22 cases reported (including his own) can be accepted into this category. The reason for this is self-explanatory when the pathology (see below) is understood.

Many different etiologic factors have been postulated although no definitive acceptable etiology has been established. In particular, tuberculosis has been implicated by investigators favoring a bacteriologic mechanism.

PATHOLOGY

The pathology and pathogenesis of sarcoidosis have been well described[6] and a summary is all that is needed here.

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The histologic findings involve all organs similarly. A mass of noncaseating tubercles, made up of large, pale, epithelioid cells having few or no surrounding lymphocytes, tends to occur in clusters about blood vessels or lymphatics and forms granulomas which rarely become confluent. Considerable fibrosis often accompanies these granulomas. Although absence of necrosis has been held to be an important differential point from tuberculous granulomas, small necrotic foci in the central portions of the lesions occasionally occur and are no longer considered incompatible with sarcoidosis. This form of necrosis rarely involves more than a few scattered lesions, is restricted to the center of the granuloma, and can usually be distinguished from the caseation seen with tuberculosis.

Analysis of 92 autopsy cases by Longcope and Freiman[6] revealed the following incidence of organ involvement: lung, 86 per cent; lymph nodes, 86 per cent; liver, 65 per cent; and spleen, 63 per cent. Less common involvement of the heart, kidney, bone marrow and pancreas occurs in that order. Sarcoid of the stomach was not mentioned and apparently was not found. Tuberculosis was associated in 19 per cent of cases.

Almost all subacute and chronic inflammatory disease processes, including tuberculosis, brucellosis, syphilis, mycotic and other infectious agents and carcinoma, have the capacity of causing pathologic reactions resembling sarcoidosis. It is sometimes impossible to determine in isolated lesions whether the pathology is due to sarcoidosis or if the sarcoid-like lesions are the result of other pathology. Considerable caution must therefore be used in reaching conclusions on histologic findings alone, and distinction between a lesion having a sarcoid appearance and the disseminated disease of sarcoidosis should be made. *The diagnosis of sarcoidosis is on firm ground only when, in addition to the proper histologic appearance, there is more than one organ involved.* It is of course possible, some hold, that the disease is merely a nonspecific reaction of the body to several different etiologic agents.[11]

Exacerbations and remissions are not uncommon and complete resolution of the pathology without remnant can occur. On the other hand, a gradual, continuing sclerosis may occur to a considerable degree. Death due to the disease itself may occur and some reports give a mortality rate as high as 25 per cent.[10] The usual cause of death is pulmonary fibrosis with associated cardiorespiratory insufficiency. Sometimes extensive involvement of the heart, kidney, or central nervous system may be fatal.[4]

In sarcoidosis of the stomach, the infiltrated gastric wall may show considerable thickening, the submucosa may be involved and there may be ulcerations and erosions. This may result in narrowing of the stomach and various degrees of stiffening of the walls. Also there may be small granulomatous involvement, which is usually present about the stomach and duodenum and may be scattered throughout the mesentery.

CLINICAL HISTORY AND FINDINGS IN GASTRIC SARCOIDOSIS

Sirak[13] presented one case of gastric sarcoidosis and reviewed 12 others taken from the literature. Nine out of 10 had absence of free hydrochloric acid. The following is a summary of the symptoms in these cases: (1) significant weight loss (8 patients); (2) epigastric distress, burning or pain (6 patients); (3) epigastric fullness, heaviness or pressure (4 patients); (4) vomiting (4 patients); (5) nausea (3 patients); and (6) gastric hemorrhage (1 patient); this was a prominent symptom in our case.

It is readily obvious that these findings are nonspecific and can occur in the presence of gastric carcinoma, peptic ulcer, cholecystitis and cholelithiasis, pancreatitis, etc.

ROENTGEN FINDINGS

The roentgen findings of gastric sarcoidosis described in the literature[2,9,11,13]

and those in our case may be summarized in the approximate order of frequency as follows: (1) antral narrowing and/or pyloric stenosis; (2) gastric ulcer; (3) infiltration of the gastric wall with stiffening and narrowing; (4) linitis plastica appearance; (5) duodenal deformity and/or ulcer; (6) abnormal gastric mucosal pattern; (7) polypoid appearance; and (8) associated abnormal small bowel appearance.

The presence of small scattered mucosal granulomas gave a polypoid appearance on the roentgenograms of the stomach in one case[2] and sarcoid tubercles were found in the gastric wall at postmortem examination in a second case. A normal appearing stomach is the most common roentgenographic finding according to Palmer's data.[8]

It is most likely that the ulcerations which occur are secondary to the infiltration, although it is conceivable that the sarcoid reaction could have resulted from the ulcer.[2] The antral and pyloric narrowing and the linitis plastica appearance are also the result of the sarcoid infiltration of the submucosa and muscle layers.

The roentgenographic appearance may therefore resemble gastric and/or duodenal ulcer. It may resemble chronic gastritis, expecially antral gastritis, linitis plastica and other causes of narrowing of the distal stomach, including syphilis, tuberculosis, fungus diseases, regional ileitis or benign hypertrophic pyloric muscle. Before sarcoidosis was recognized as a clinical entity, cases of gastric sarcoidosis were classified as tuberculosis.[2]

In the absence of any signs of sarcoidosis elsewhere in the body, the possibility of gastric sarcoidosis should receive at most only secondary consideration. Benign hypertrophic pyloric muscle gives a fairly typical appearance which usually can be recognized. Whenever a regional ileitis has involved the stomach, there has been evidence of associated involvement somewhere in the small intestine. Syphilis of the stomach in the present day has become extremely rare; however, in the face of positive serology, this etiology must receive consideration and may have to be excluded by therapeutic tests. Although gastric carcinoma can rarely extend across the pylorus and involve the duodenal bulb, the presence of such extension usually suggests chronic duodenal and gastric inflammation secondary to peptic ulcers.

It can be seen that the roentgenographic diagnosis of sarcoidosis is impossible to establish since it resembles other disease entities which are much more common and others which are more rare. However, the important consideration to be remembered is that, in the presence of sarcoidosis elsewhere in the body, evidence of gastric and duodenal pathology must bring to mind the possibility of sarcoidosis of the upper gastrointestinal tract.

There has been only one case of sarcoidosis of the esophagus reported, according to Allen, Batten and Jefferson,[2] and this showed only a localized stricture. An abnormal mucosal pattern may occur in the small intestine by virtue of mesenteric involvement but without actual involvement of the wall. On the other hand, actual infiltration giving the mucosa a swollen, hypertrophic appearance and polypoid filling defects (considered to be due to mucosal epithelial tubercles) have been reported.[14] The incidence of small bowel sarcoidosis is apparently even less than that in gastric sarcoidosis. There have been only 4 cases of colon sarcoidosis reported.[1]

THERAPY

Cortisone administration results in both subjective and objective improvement of varying degrees in most patients and, where ulcer symptoms or roentgenographic signs are present, the usual medical management of peptic ulcer should be instituted. Patients in whom cortisone therapy is terminated usually undergo relapse.[13] Surgical intervention is indicated for the complications of gastric sarcoidosis (in cases where hemorrhage cannot be controlled or is recurrent, or there is obstruction, intractable pain and failure of the ulcer to heal). Although gastric sarcoidosis

may produce ulceration, one must be cognizant of gastric ulceration secondary to cortisone therapy *per se*.

REPORT OF A CASE

L.T., a thirty-eight year old Negro female, had had three previous hospital admissions elsewhere over a four year period, the last two years prior to admission to this hospital. Her first hospitalization was in June, 1953, at which time she complained of abdominal swelling associated with occasional nausea and vomiting for a period of one year. A chest roentgenogram revealed evidence of extensive inflammatory lesions scattered about the right hilus with some involvement of the left lower lung field. Hepatosplenomegaly, as well as generalized lymphadenopathy, was present. Liver function was impaired, as indicated by a thymol turbidity of 14.2 units and a cephalin flocculation of 4+, and the total serum proteins ranged from 6.1 to 9.2 gm./100 cc. with a reversal of the A/G ratio. Biopsies of the skin, axillary lymph node and liver were diagnostic of Boeck's sarcoid. She was given a short period of cortisone therapy with relief of symptoms.

FIG. 1. Prepyloric ulcer with fixation of gastric wall in the prepyloric region which was considerably narrowed. Delayed gastric emptying was associated.

The patient's second hospital admission was in June, 1955, because of a six month history of amenorrhea, urgency of urination and constiptation. The liver and spleen were again noted to be enlarged and a chest roentgenogram revealed some regression of the inflammatory process previously observed. She was found to have prolapse of the uterus associated with a cystocele and a rectocele. No operation was performed and she was again treated with metacorten (5 mg. every eight hours), this time for a period of three months following dismissal from the hospital.

The third hospitalization was in May, 1956, because of an episode of hematemesis and melena without symptoms of peptic ulceration. The liver and spleen were again noted to be enlarged and the hemoglobin was 6 gm. on admission. An upper gastrointestinal series revealed partial obstruction of the pylorus; gastroscopy showed marked hyperemia of the gastric mucosa, and a gastric analysis was negative for free acid. The patient had several episodes of hematemesis and melena over the first week of hospitalization, necessitating ten units of blood; however, this was finally controlled on medical management and she was discharged on antacid therapy.

On the fourth and last admission, this time at Jefferson Davis Hospital, the patient gave a history of intermittent abdominal swelling with excessive gas, weakness and vomiting during the two years since the previous admission. All symptoms had become gradually more intense, but there was no pain. Associated weight loss was noted, the abdomen was distended and tympanitic and the liver was enlarged but without associated splenomegaly. No history of hematemesis or melena was elicited, but the hemoglobin was 5.4 gm., red blood cell count 2.9 m. per cu. mm. and the hematocrit was 28 per cent. The gastric analysis revealed 150° of free hydrochloric acid and 300° total acidity.

The roentgenogram of the upper gastrointestinal tract demonstrated moderate delay in gastric emptying, marked narrowing of the prepyloric area, and fixation of the gastric wall in this region, associated with a prepyloric ulcer and an ulcer on the lesser curvature margin of the pars media (Fig. 1). The duodenal bulb was deformed, largely by extrinsic pres-

sure, and there was hypertrophy of the mucosal pattern in the first portion of the duodenum (Fig. 2). The esophagus appeared very slightly dilated with a heavy mucosal pattern but, on subsequent postoperative examination one month later, it appeared normal. The patient had not been treated prior to admission or in the meantime with hydrocortisone or any other medications thought to be of specific value in this disease. There was some disturbance of the usual small bowel appearance with slight relative widening of two or three loops of jejunum and some loss of the usual mucosal pattern (Fig. 3). The fibrotic infiltration in the right perihilar region had undergone no change over a period of several years, indicating an inactive pulmonary process.

The patient continued to have intermittent abdominal distention and a subtotal gastrectomy of the Billroth II type was performed after she had been transfused with whole blood. An ulcer, 0.3×0.4 cm., was found 8 cm. proximal to the pylorus on the lesser curvature margin of the stomach and this was surrounded by an area of thickening and induration. On the posterior gastric wall and in the duodenum were scattered superficial mucosal erosions, some measuring up to 1.5 cm. in diameter. Attached to the resected stomach were numerous lymph nodes, gray-pink in color, measuring 1.5 to 2 cm. in diameter. Enlarged mesenteric lymph nodes were also present and some of these caused extrinsic pressure on the first portion of the duodenum. No involvement of the bowel wall was noted (other than the first portion of the duodenum) and no biopsy was taken from the small intestine or colon.

The microscopic examination revealed the defects in the gastric mucosa and thickening of the submucosa and muscle layers by fibrous connective tissue which contained several granulomata made up of epithelioid cells, surrounded by lymphocytes and fibroblasts. Occasional multinucleated giant cells were present. There was no evidence of caseation. Similar granulomata were seen in numerous areas of the stomach and duodenum, including the tunica propria, submucosa, and muscle layers. Other granulomata were present in the peritoneal lymph nodes. Acid-fast and period acid Schiff stains revealed no organisms.

The pathologic diagnosis was Boeck's sarcoidosis involving the stomach, duodenum, lymph nodes, peritoneum and liver, with associated gastric ulcer, chronic and acute, and superficial gastric and duodenal mucosal erosion. The mesenteric lymph node involvement can account for the roentgenographic changes in the small intestine; these changes may be explained by irritation of the nerves, vascular spasm or by deranged flow of lymph as suggested by Golden.[5]

Fig. 2. Deformed duodenal bulb due largely to extrinsic pressure by enlarged lymph nodes. The mucosal pattern is thickened.

CONCLUSION

Sarcoidosis of the stomach is a rarity although pathologic involvement of the stomach in one series has been shown to occur in approximately 10 per cent of the cases having no gastrointestinal symptoms or roentgenographic signs.[8] In most cases

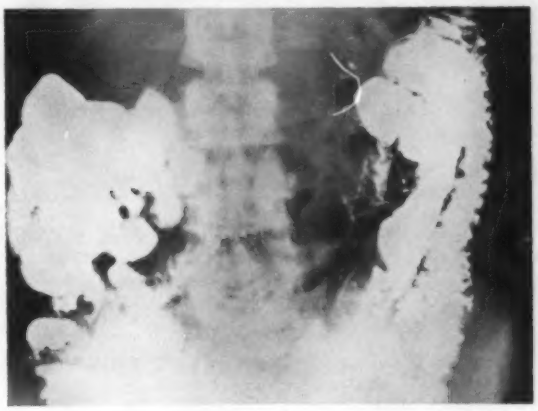

Fig. 3. Dilatation and mucosal thickening in proximal jejunum.

exhibiting roentgenographic evidence of gastric pathology, symptoms suggestive of peptic ulcer are present.[13] It, therefore, seems likely that a larger number of patients with sarcoidosis elsewhere and gastrointestinal symptomatology should be found to have positive pathologic and roentgenologic evidence of gastric sarcoidosis.

It is important that the radiologist, aware of the clinical and pathologic diagnosis of sarcoidosis in a patient, should search the stomach and duodenum carefully for evidence of pathology.

The most common roentgenographic findings in cases of this disease are antral and pyloric stiffening and narrowing, gastric and duodenal ulcers and deformity of these regions, and disturbance of the mucosal pattern.

In the presence of positive roentgenographic findings, gastroscopy and biopsy are suggested to confirm the possibility of gastric sarcoidosis.

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UTERINE LEIOMYOMA AS A CAUSE OF SMALL BOWEL OBSTRUCTION*

A CASE REPORT OF TUMOR ERODING THE INTESTINE AND FLAKING CALCIFICATION INTO THE BOWEL LUMEN

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THIS report deals with a calcified uterine leiomyoma which eroded the t rminal ileum and caused partial obstruction. The diagnosis was made from roentgenograms preoperatively because flaked calcifications were seen in the small bowel proximal and distal to the point of obstruction. Prior to searching the literature, we expected that the unique feature of this case would be the calcification free within the bowel lumen. Much to our surprise we found no case in which a benign tumor had caused obstruction after entering through the intestinal wall. There are, of course, many examples of leiomyomata which have caused small and large bowel obstruction by extrinsic pressure.[2,3]

Thus two features, benign tumor erosion into the bowel causing obstruction and free calcification in the bowel lumen, prompt us to report this unusual case.

REPORT OF A CASE

This sixty-one year old Negro female was admitted to University Hospital with a chief complaint of profuse vomiting of one week's duration. She gave a history of intermittent episodes of cramping left lower quadrant abdominal pain and a 50 pound weight loss during the previous two months. There had been no change in bowel habits and no melena. Examination on admission revealed an acutely ill patient whose temperature was 98.4° F., pulse 76 per minute, respirations 22 per minute, and blood pressure 140/78 mm. Hg. Her abdomen was tympanitic with hyperactive bowel sounds and peristaltic rushes over the lower half. Gynecologic examination demonstrated an irregularly enlarged uterus with a large anterior mass thought to be a leiomyoma. No other organs or masses were palpable.

Laboratory Studies. The hemoglobin was 8.2 gm.; leukocyte count 10,600; carbon dioxide 33 mEq.; chloride 91 mEq.

Roentgenologic Examination. A roentgenogram of the abdomen (Fig. 1) on the second hospital day revealed a pattern of partial large bowel obstruction. In addition to calcification typical of uterine leiomyomata, there were

FIG. 1. Plain roentgenogram of the abdomen on second hospital day. Two areas of calcification typical of uterine leiomyomata overlie the sacral area. Also note the flaked opacities along the left abdomen which appear to be in the descending colon (see Figure 2).

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FIG. 2. Barium enema evacuation roentgenogram made approximately one hour after that in Figure 1. The opacities are seen to be within small bowel medial to the partially empty descending colon.

amorphous calcific densities which appeared to be mixed with fecal contents in the descending colon. A barium enema study made the same day, however, showed that the abnormal calcifications were not within the colon but instead were within the small bowel (Fig. 2). Review of a roentgenogram made twenty-four hours previously did not show the flaked calcifications and no radiopaque media or medication had been given in the interim.

Course. The patient was treated with appropriate intravenous fluids and gastric decompression prior to surgery for intestinal obstruction. On the twelfth hospital day an exploratory laparotomy was performed. An obstruction of the terminal ileum was found some 20 cm. from the cecum. The ileum was adherent to a calcified uterine leiomyoma at the point of obstruction. A segmental resection of the involved small bowel plus a total hysterectomy was performed. The postoperative course was uneventful.

Pathology. The ileum was opened longitudinally and is shown on a roentgenogram (Fig. 3) and a photograph of the gross specimen (Fig. 4). As can be seen, the tumor had eroded the intestinal wall and was occupying most of the lumen. That segment of the tumor within the lumen clearly contained calcium, and flecks were seen scattered in the intestinal contents. On microscopic examination the lesion was declared to be a simple leiomyoma without evidence of malignancy.

DISCUSSION

Based on the roentgenologic findings it was postulated preoperatively that sarcomatous degeneration of a leiomyoma with bowel invasion would explain the apparent relationship between the calcified uterine fibroid, small bowel obstruction and intraluminal calcifications. However, as indicated above under "Pathology" no evidence of malignancy was found.

It is not possible to detail the exact mechanism by which this erosion of a benign tumor into the small bowel occurred. The following possibilities exist: (1) The calcified surface of the tumor irritated the bowel wall with eventual erosion. (2) Adhesions united the leiomyoma with the bowel wall. As the myoma grew, the bowel wall was put on a stretch and with compromise of the wall's blood supply, necrosis occurred and the tumor entered.

We favor the second explanation of adhesions and eventual erosion. This mechanism would be quite similar to that resulting in the so-called "parasitic" leiomyomata. Here the tumor becomes attached to the bowel wall and gradually gains more of its blood supply from the vessels supplying the intestine, until eventually the leiomyoma may become completely detached from the uterus. The point that interests us here, however, is that there are reported cases in which the leiomyoma communicated with the bowel lumen and became "hollowed out" with free entrance and egress of intestinal contents.[1] If we go back one step in such a case, to the point when the center of the tumor was being evacuated into the bowel lumen, then we duplicate the appearance in our specimen. If one recalls the manner in which a leio-

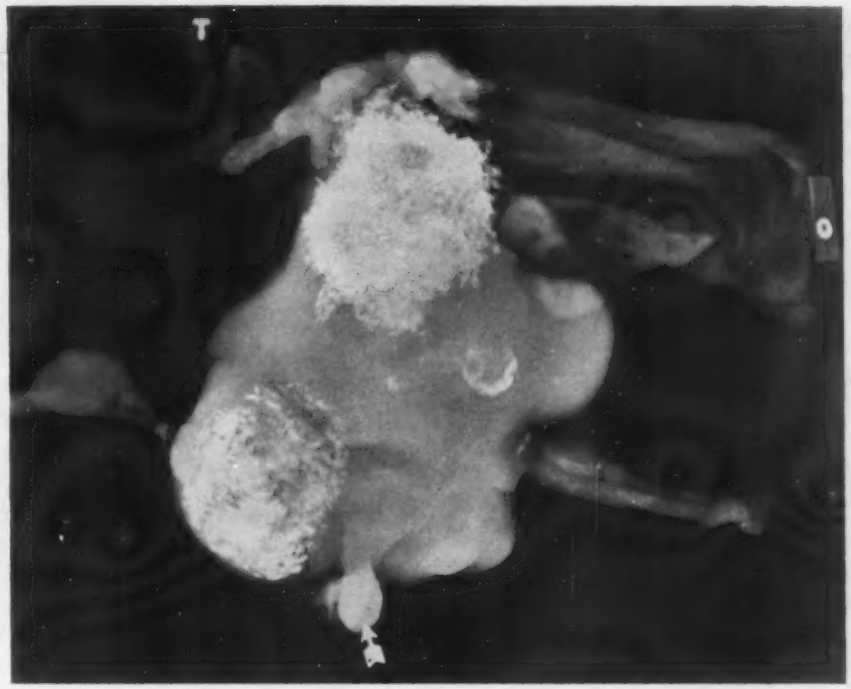

Fig. 3. Roentgenogram of the surgical specimen. The arrow below points to a distorted cervix. The calcifications identify leiomyomata in the lobulated, enlarged uterus. The ileum is seen above with "T" representing the terminal portion while "O" identifies the opening of the proximal portion. The orientation of the specimen corresponds with that shown in Figures 1 and 2. Above the upper calcified leiomyoma, the ideal wall is thickened and therefore casts a more opaque shadow. This point is the actual site of entry of the tumor into the bowel.

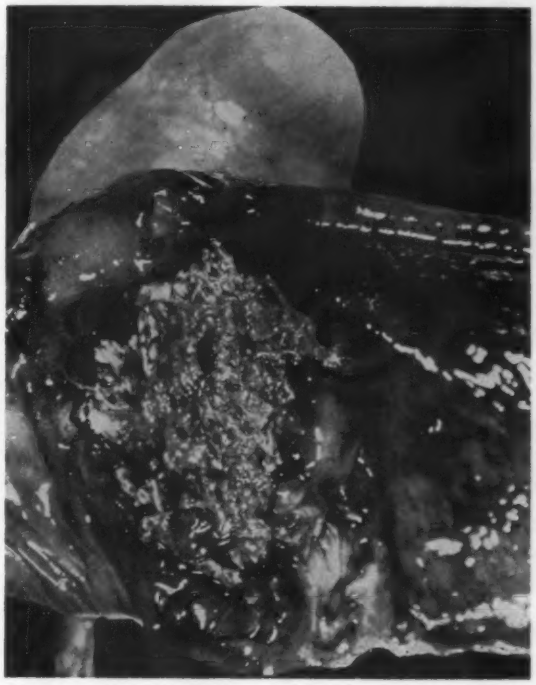

Fig. 4. Photograph of the surgical specimen with the ileum opened longitudinally; it is shown from above in order to best see that portion of the leiomyoma within the bowel lumen. The whitish mass seen over the opened ileum is one of the uterine lobulations.

myoma tends to extrude its interior when its surface is cut, this hypothesis becomes tenable.

The ileum was involved in an area where a Meckel's diverticulum might have existed. If present, diverticulitis may have served as the inciting agent for adhesions to form between the bowel wall and the leiomyoma.

SUMMARY

We have presented a unique case in which a calcified leiomyoma entered the distal ileum and caused partial obstruction. The diagnosis was made preoperatively due to the presence within the small bowel of calcific flakes which were correctly considered to have come from the leiomyoma. The possible mechanisms of occurrence are discussed.

The combination of calcified leiomyomata and intestinal obstruction should raise in the radiologist's mind the possibility of the former causing the latter, particularly if there are otherwise unexplained opacities within the bowel lumen.

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Vol. 84, No. 2

A REVIEW OF ROENTGEN SIGNS IN LESIONS AFFECTING THE CECUM

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IT IS common knowledge that certain roentgen findings in lesions of the cecum, as in lesions elsewhere in the body, are fairly typical of some specific disease so that a "working diagnosis" can often be made which is satisfactory for the initial management of the patient. A description of a roentgen finding such as a *filling defect* is, however, in many cases no more a definitive diagnosis than is a description of a physical finding such as a *mass*. The language of radiology is not ideally suited for describing roentgen findings because it includes a conglomeration of terms borrowed from other branches of medicine such as anatomy, pathology and clinical medicine. These terms are descriptive and are subject to nuances of definitions or interpretations which are often subconsciously colored by experience, knowledge of previous studies or the clinical history of the patient. Furthermore, the pathologic process is frequently implied in the descriptive roentgen term used; as in "contracted cecum," "ulcerative contour," and "annular neoplasm."

DESCRIPTION OF ROENTGEN SIGNS

Table 1 lists the signs or findings which will be used as a frame of reference though not as criteria since definite standards do not exist. It should be understood that some of these roentgen findings do not apply in many conditions and that most lesions exhibit several of these signs with varying degrees of significance. For purposes of this presentation, the roentgen signs used are the ones which seem most definitive, although the same lesion might be described by other terms, for example, "filling defects" might also be described in

TABLE I

ROENTGEN SIGNS IN LESIONS AFFECTING THE CECUM

Morphology.	
Caliber	Narrowing Dilatation
Protrusion	Intrusion (filling defect) Extrusion (pouch,[8] fistula, channel or niche)
Margin	Irregularity Smoothness
Mobility	Fixation Hypermobility
Length	Shortening Elongation
Relief Pattern	Distortion Obliteration
Position	Malposition[29] Displacement[30]
Functional Motor Activity.	
Filling and Emptying	Spasticity (Stierlin's sign) Irritability
	Rapid emptying Nonfilling
	Hypotonicity Stasis Overfilling

ASSOCIATED FINDINGS COMMON TO SEVERAL ROENTGEN SIGNS

1. Extent of lesion
2. Site of lesion: ileocecal junction, tip or body of cecum or appendiceal zone
3. Roentgen findings in adjacent or distant structures
4. Modification of signs by special procedures
5. Roentgenographic evolution of the process: stationary, or rate of progression or regression

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terms of the resultant "narrowing" of the lumen. The descriptive roentgen morphologic signs listed in the table are self explanatory.

The functional motor activity of the cecum, as elsewhere in the gastrointestinal tract, usually plays a secondary or minor role, from a radiologic viewpoint. This is because of the absence of valid roentgenographic criteria of function which often leads to unreliable control studies and premature correlation between the roentgenographic findings and the clinical aspects of the disease, although in advanced cases satisfactory correlation can be established. A mountain of information and opinion, incomplete and unsubstantiated, exists in relation to the field of functional activity of the gastrointestinal tract, from the standpoint of radiology. Further study is necessary to establish the proper balance between the two extreme schools of roentgen diagnosis: the one that relegates the functional factors to zero and depends only on obvious organic lesions, and the other that tends to base every clinical symptom or finding on some observed functional variation of the alimentary tract in the absence of definite morphologic changes. It should be emphasized that the border zone from normal to abnormal, both anatomically and physiologically, is very broad and and gradual rather than sharply defined (Fig. 4*A*, 12*A*, 13*A* and 15*A*).

Peristaltic activity, an important functional motor activity of the remainder of the gastrointestinal tract, appears to play an insignificant role in the cecum. The term "tonus" as here used is an expression of the degree of muscle activity of the cecum as demonstrated roentgenologically. Filling and emptying of the cecum is obviously related to tonus, but it also implies an additional factor not expressed by tonus; namely, the process of opaque material entering the cecum either from the ileum or in retrograde fashion from the colon. "Transit time" is another term used in describing the filling and emptying of the cecum, by way of the ileum, and progression of its contents into the ascending colon.

Roentgenologic study of the cecum and the pericecal area is accomplished by one or several of the following procedures: survey roentgenogram of the abdomen, single contrast enema, double contrast study[25,36] and oral opaque meal. Often various techniques and maneuvers (Fig. 1) are necessary to fully evaluate the region; such as the use of high kilovoltage,[17] special barium preparations, tannic acid,[7] water soluble opaque preparations, and spot roentgenograms,[14] with and without pressure. The procedures, techniques and maneuvers to be employed must be decided by the roentgenologist for each presenting case. Thorough preparation of the patient is of prime importance for the single and double contrast studies; otherwise, the study is likely to be a failure or even misleading.

ROENTGEN SIGNS AS SEEN ON SURVEY ROENTGENOGRAMS

While the roentgen signs listed in Table 1 apply primarily to opaque contrast studies, with some limitations they may also be applied to certain situations on survey roentgenograms of the abdomen. On these, since gas is a contrasting medium, some idea as to the size, configuration and position of the cecum may be obtained. Limiting factors in the interpretation of the survey roentgenogram, however, are the extreme variability in the amount of gas present in the intestinal tract and the inferior contrast produced by gas as compared with opaque material.

The use of decompression tubes, cleansing enemas, or drugs affecting the intestinal musculature modifies the picture and thus may aid in proper interpretation. The signs may also be modified by changing the position of the patient; this is particularly useful in the detection of fluid levels. In acute abdominal conditions survey roentgenograms of the abdomen often are the only studies possible and the most should be made of them. From the foregoing it should be apparent that the survey roent-

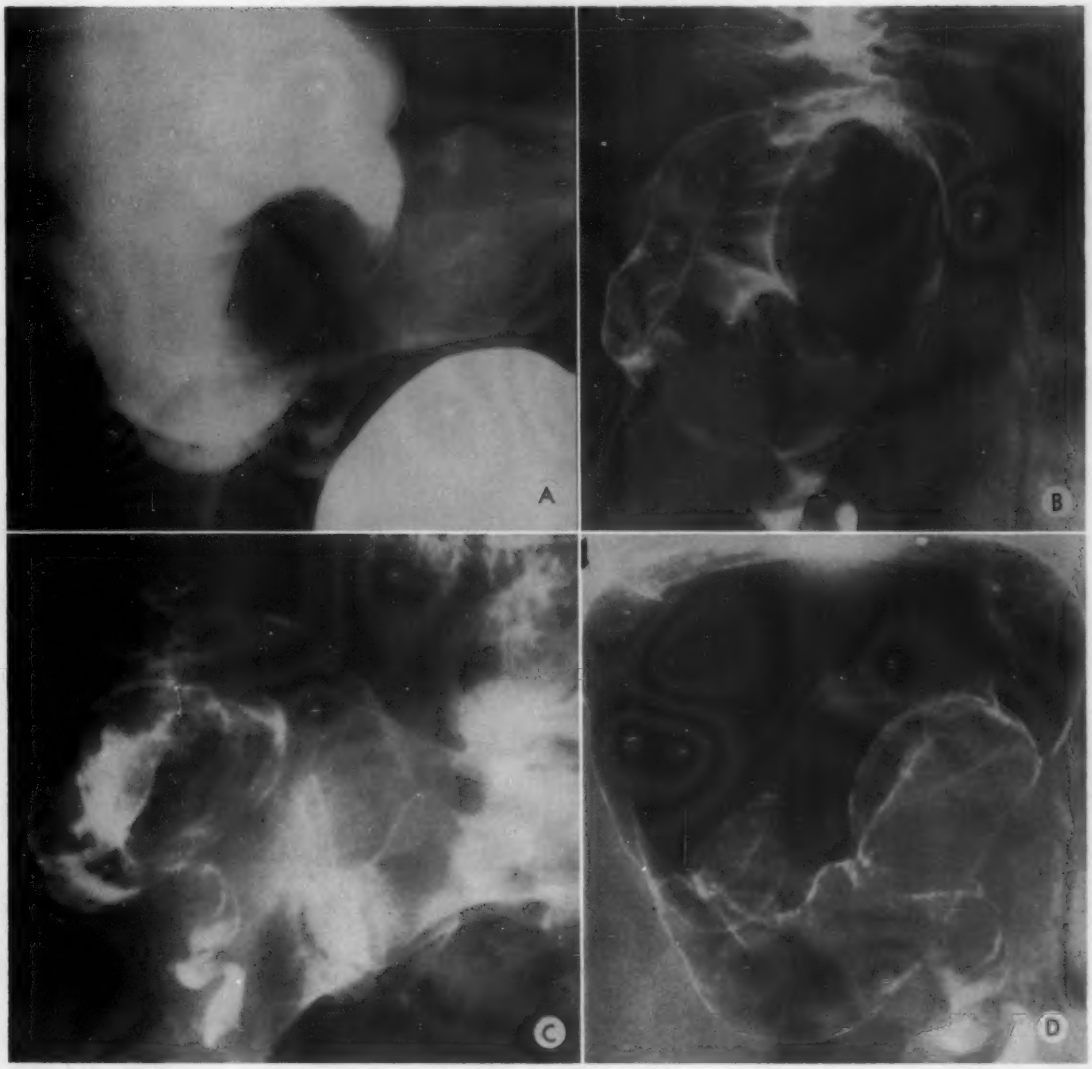

Fig. 1. *Various roentgen manifestations of the same lesion with different techniques.* Pathologic diagnosis: carcinoma of cecum. (*A*) Routine barium enema roentgenogram—filling defect of cecum with smooth contour. (*B*) Postevacuation roentgenogram with pressure—obliteration of mucosal pattern over filling defect. (*C*) Postevacuation roentgenogram—mild intussusception with filling defect. (*D*) Double contrast roentgenogram—polypoid filling defect with lobulated surface contour.

genograms are often equivocal or noncontributory, but at times are suggestive of certain abnormal states. The following points should be looked for:

Paucity or Absence of Gas (Fig. 2). Normally, there is a sufficient amount of gas and fecal matter within the cecum to be identified as such. Paucity or lack of gas and fecal matter in the cecum, associated with gas in the adjacent ileum and colon, is sometimes demonstrated in acute appendiceal disease.[46]

Localized Haziness. Localized haziness, poorly or sharply defined, in conjunction with the above findings is occasionally seen in mass lesions of the cecum or pericecal tissues, and these may be inflammatory or neoplastic in nature (Fig. 2, *C* and *D*). In infants and children this sign, particularly when coupled with a mass density and if

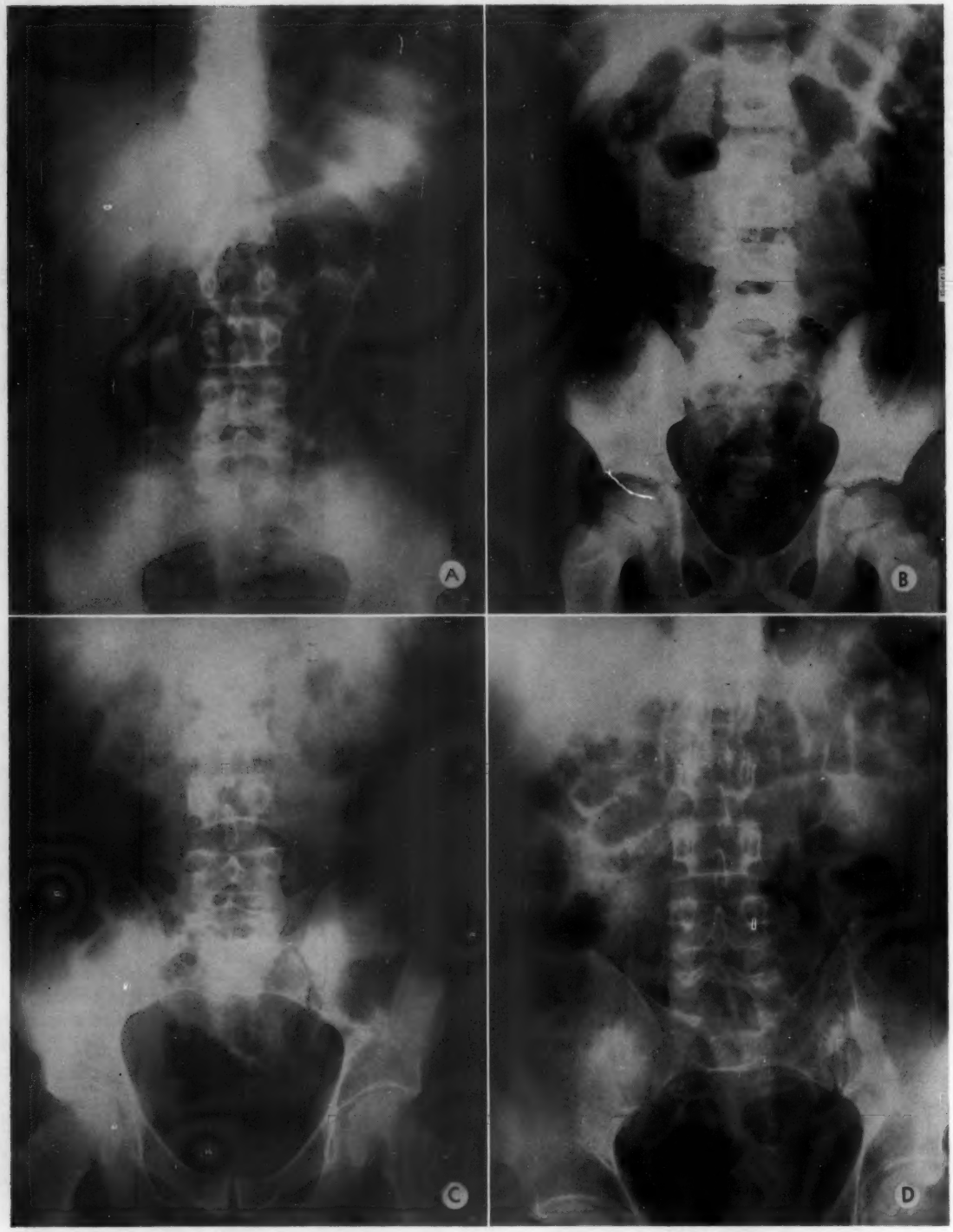

FIG. 2. *Paucity or absence of gas in the cecum with gas in the adjacent segments of small and large intestines.* (*A*) Operative findings: acute appendicitis. (*B*) Operative findings: ruptured appendix. (*C*) Operative findings: appendiceal abscess. (*D*) Operative and pathologic findings: carcinoma of cecum associated with pericecal inflammation.

associated with adequate history, may confirm a clinical impression of intussusception. In exceptional cases the cecum in volvulus or in closed loop obstruction may be filled with fluid and masquerade as a large mass density (Fig. 3*D*). Rarely, a fine granular "air-bubble tapioca" pattern is seen within the haziness which may suggest abscess formation.

Localized Collection of a Large Amount of Gas (Fig. 3, *A*, *B* and *C*; and 4, *B* and *D*). A marked amount of gas localized in the region of the normally situated cecum or, by its configuration, highly suggestive of a misplaced cecum (Fig. 3*A*, and 4*B*) is another important finding on the survey roentgenogram. Although the cecum is the most dilatable segment of the colon, it has the thinnest walls and perforation is likely to occur when it is distended beyond a "critical" size. Acute perforation of the cecum carries with it a high rate of mortality and it is therefore imperative to recognize the markedly dilated cecum as early as possible, since this finding is decisive in suggesting immediate surgical intervention. Davis and Lowman[10] have estimated the critical size of the cecum to be 9 cm. in diameter for impending perforation (Fig. 3*D* and 4*D*). This is a useful but crude guide since rupture, in addition to size, may depend on the speed of development, the presence or absence of distention of the adjacent small intestine and probably other factors not as yet well understood. Multiple perforations of the cecum may occur without revealing their presence on a roentgenogram (Fig. 4*C*).

The cecum, being a blind pouch, will respond to obstructing lesions distal to it, in a manner dependent on the function of the ileocecal valve. The etymologic meaning of "competence" or "incompetence" of the ileocecal valve is confusing and these terms will not be used here. However, in cases of obstruction distal to the cecocolic junction, the cecum apparently dilates if the valve allows a greater inflow of small intestinal contents into the cecum than a backflow from the cecum into the small intestine. The nature and location of the lesion causing the marked dilatation of the cecum may not be apparent or definite on the survey roentgenogram and if the situation permits, this may be determined by an opaque contrast enema study. In adults 90 per cent of mechanical obstruction of the colon is due to carcinoma. Other causes include diverticulitis, extrinsic neoplastic growths, inflammatory masses, fecal impactions and incarcerated hernia.[2] Spontaneous perforation[35] of the markedly dilated cecum has been reported in the absence of obvious mechanical obstruction of the colon.

When an unusually situated, large collection of intestinal gas is suspected of being in the cecum, then volvulus of the cecum[1] and ascending colon must be considered (Fig. 3, *A* and *C*; and 4*B*). The severity of the volvulus depends on the degree or grade of torsion of the mobile cecum with its long, loose mesentery. With progression of the torsion beyond 180 degrees, not only does a closed-loop obstruction of the cecum develop, but a progressive gaseous distention of the small intestine occurs, within twelve to twenty-four hours, as a result of the mechanical obstruction of the ileum. Fluid levels are found both in the small intestine and in the dilated cecum. At times several levels may be found in the cecum and ascending colon, due to their large size, disposition and lie. Although there is no one specific accessory activating factor, 50 per cent of the cases in one study were found to be associated with other lesions of the distal colon.[34] Haziness in the lesser pelvis coupled with the above findings suggests free fluid in the pelvis or loops of small intestine distended with fluid.

The cecum may also dilate as part of a more generalized dilatation of the intestine, such as occurs in mesenteric vessel obstruction or paralytic ileus, although the degree of distention of the cecum in these cases is not as marked as in mechanical or closed loop obstruction of the cecum (Fig. 3*B*).

Calcification. Calcification may be found

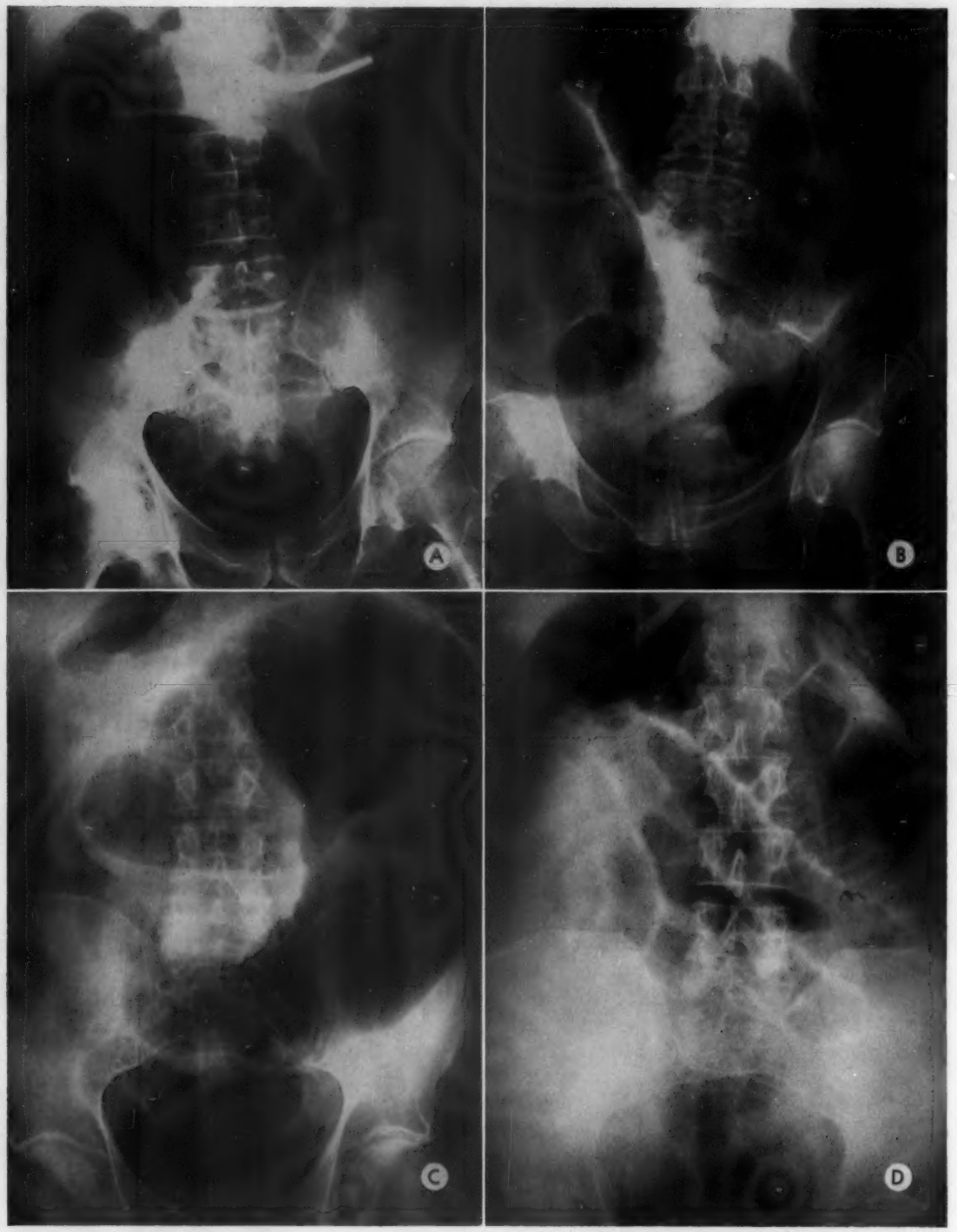

FIG. 3. *Cecum markedly dilated with air and/or fluid.* (*A*) Roentgen findings: cecum dilated to "critical" size and cecum in abnormal position; no air in small bowel. Operative findings: volvulus of cecum. (*B*) Roentgen findings: marked dilatation of cecum and colon to sigmoid region; no air in small bowel. Operative findings: annular carcinoma of sigmoid; closed loop obstruction of cecum. (*C*) Roentgen findings: markedly dilated cecum; cecum abnormal in position; air in small bowel. Operative findings: volvulus of cecum. (*D*) Roentgen findings: homogeneous mass density on right side of abdomen; air in small bowel. Operative findings: carcinoma of hepatic flexure; markedly dilated cecum filled with fluid with impending perforations of cecum.

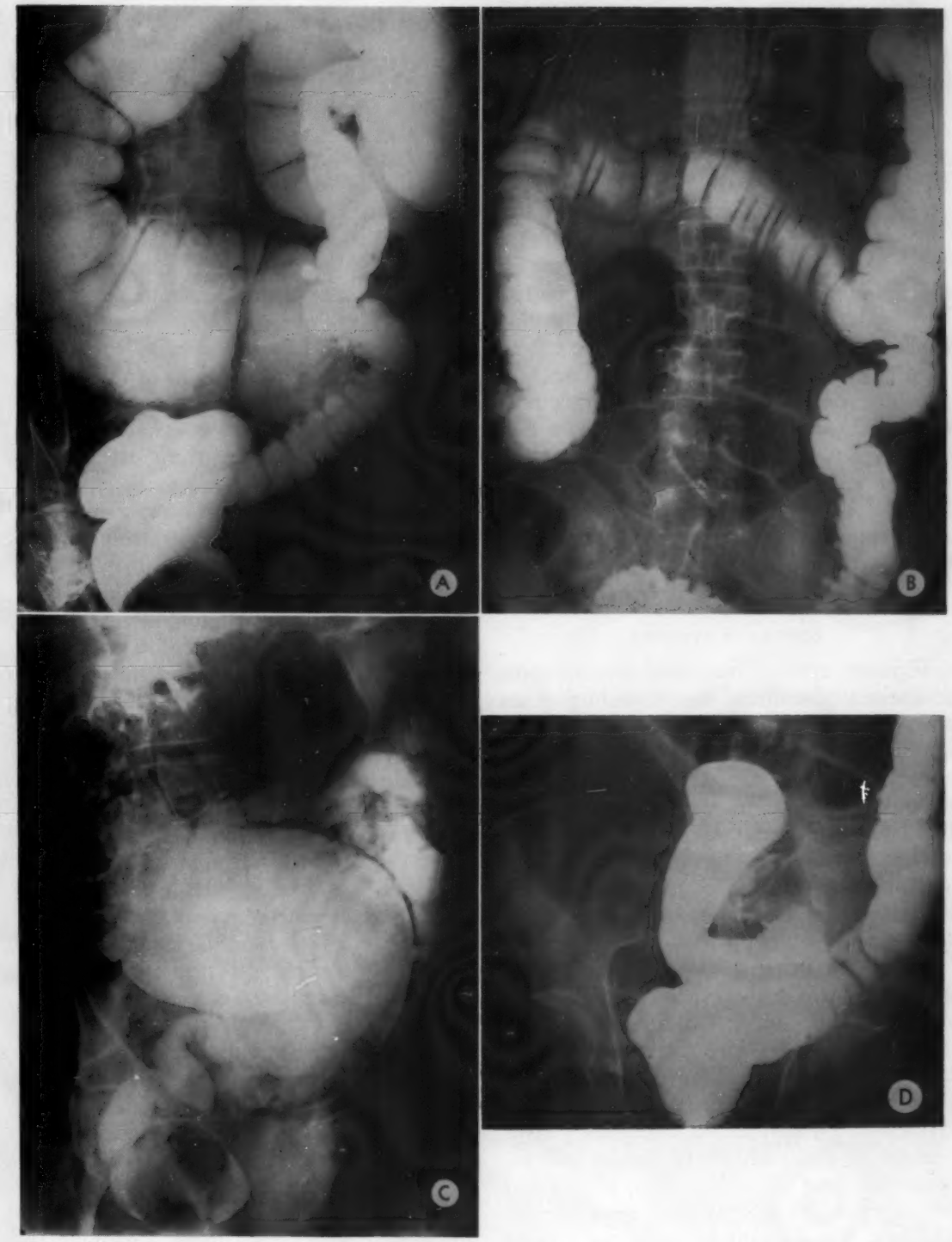

FIG. 4. *Cecum dilated with air or barium.* (*A*) Operative findings: dilated cecum and proximal colon; no obstruction. (*B*) Roentgen findings: volvulus of the cecum with gaseous distention of adjacent small intestine. Spontaneous detorsion of the volvulus[13] occurred subsequent to the enema examination. Operative findings (several days later): perforation of diverticula of sigmoid, and a hypermobile cecum was revealed. (*C*) Roentgen findings: retrograde obstruction of barium at hepatic flexure. Operative findings: annular carcinoma of proximal portion of transverse colon with marked dilatation of the cecum and multiple cecal perforations. (*D*) Operative findings: annular carcinoma of hepatic flexure with marked dilatation of cecum, ascending colon and small bowel; impending perforations of cecum.

in appendiceal enteroliths[4] but these are quite rare. Differential diagnosis should include right ureteral calculus, calcified lymph node and the rarer calcified gallstone which has perforated and lodged in the ileocecal region. In the latter case, air in the biliary tree may be diagnostic. Although calcified appendiceal enteroliths may be asymptomatic, often they are associated with subsequent appendiceal disease including perforation or abscess. The enterolith may have a laminated appearance. Calcification may also occur in a rare mucocele, either within the mass or as a fine calcific rim. If the situation permits, a barium enema will reveal a more exact location of the calcified density and may also demonstrate an irregularity or defect on the cecal contour.

ROENTGEN SIGNS AS SEEN ON CONTRAST STUDIES

Figures 5 through 10 are schematic representations of some of the more important roentgen signs as demonstrated by the barium filled lumen.* It is not practical to consider in detail the complete differential diagnosis of each figure; only certain of the highlights which may apply to one or more of the illustrations will be discussed.

Carcinoma is the most common lesion in the group of filling defects.[5] A filling defect with an irregular contour is more suggestive of a malignant than a benign tumor. Either irregular or smooth contours may be associated with inflammatory masses or granulomatous lesions. Alteration or distortion of the normal mucosal pattern in the cecum, as in other parts of the gastrointestinal tract, also favors malignancy over benignity, although there again, inflammatory lesions with ulceration, edema and granulomatous aspects may give similar findings. A malignant lesion is more likely than a granulomatous lesion to have a sharp transition from pathologic to normal mucosa (Fig 1, 5, 6, 11 and 12). It is not an unusual occurrence for carcinoma of the cecum and appendicitis to coexist and in such cases the carcinoma may be missed clinically and even at operation.[28] It should be stressed, therefore, that in postoperative cases of poor healing or fistula formation, roentgen studies of the cecum should be made for evidence of carcinoma.

Malignant lymphomas of the cecum[32,41] are relatively rare and comprise less than 1 per cent of malignant lesions of the bowel. Despite their occurrence in the younger age groups, malignant lymphomas cannot, as a rule, be differentiated from carcinoma, since carcinoma may occur as early as the second decade. Malignant lymphomas, apparently limited to the gastrointestinal tract, are almost invariably multiple, and may involve other segments of the bowel. While this multiplicity may be a differential point, it should be kept in mind that multiple simultaneous carcinomas of the colon do occur in a very small percentage of cases, but that these do not involve the small intestine.

Carcinoids of the cecum,[29] often malignant, may cause a filling defect with relatively smooth edges simulating a benign filling defect. They are frequently submucosal and are covered with intact mucosa. Carcinoids, especially malignant cases with hepatic metastases, may be associated with a clinical syndrome,[31] principally manifested by peculiar cutaneous flushes, chronic diarrhea, respiratory distress and late development of valvular disease of the heart—usually pulmonic stenosis and tricuspid insufficiency. It is considered that this syndrome is the result of excessive formation of a serotonin metabolite, which may be found in the urine by special tests.

Benign tumors (Fig. 12*C*),[38] including adenomatous polyps, lipoma, endometrioma, leiomyoma,[23] and enterocytoma, are very rare tumors of the cecum. Lipomas are almost all submucous[11] and most of them are solitary. They involve the ileocecal valve (Fig. 13*C*) or cecum more commonly than the remainder of the bowel.

* These are composite drawings obtained from studying hundreds of illustrations and actual roentgenograms from the authors' files and from the literature. They are not intended to be all inclusive but are presented to serve as guides.

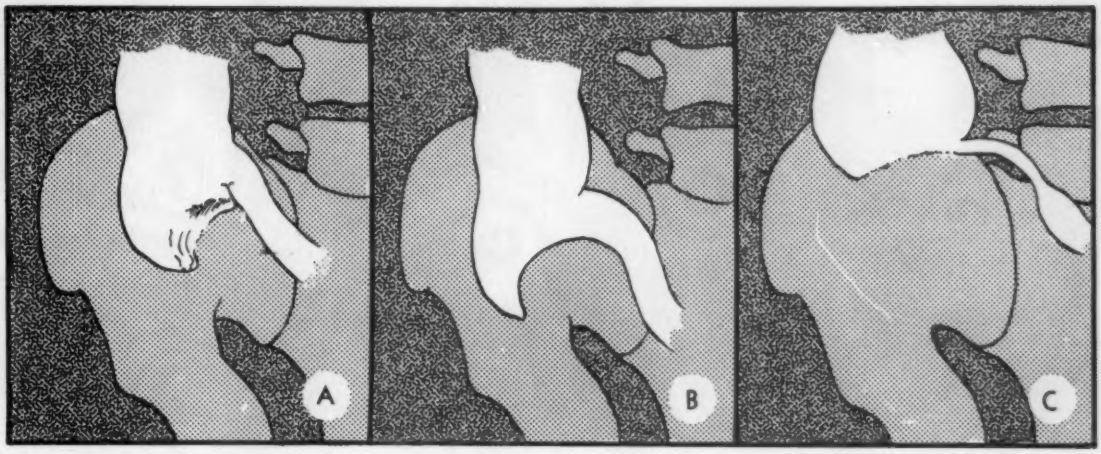

Fig. 5. *Local wall displacement and/or filling defect.* (*A*) Irregular margin. (*B*) Smooth margin; ileum displaced. (*C*) Irregular margin; ileum displaced and narrowed.
Appendiceal mass lesions: abscess, mucocele, carcinoid, carcinoma.
Cecal benign neoplasms: adenoma, leiomyoma, fibroma, carcinoid, lipoma.
Cecal malignant neoplasms: carcinoma, lymphosarcoma.
Cecal granulomatous masses: amebic granuloma, actinomycosis, hyperplastic tuberculosis.
Other extrinsic or extramucosal masses: ovarian, uterine, sigmoid colon, congenital cysts.
Postoperative defects.

They generally occur in later years of life and intussusception is the first clinical event in nearly one-half of the cases. Leiomyomas are not as common in the cecum as in the small intestine. When these tumors project into the lumen, the overlying mucosa frequently erodes and a deep ulcer is likely to be excavated from which very severe and sometimes exsanguinating hemorrhage may occur.

Normal filling of the appendix in the presence of roentgen findings of a cecal or

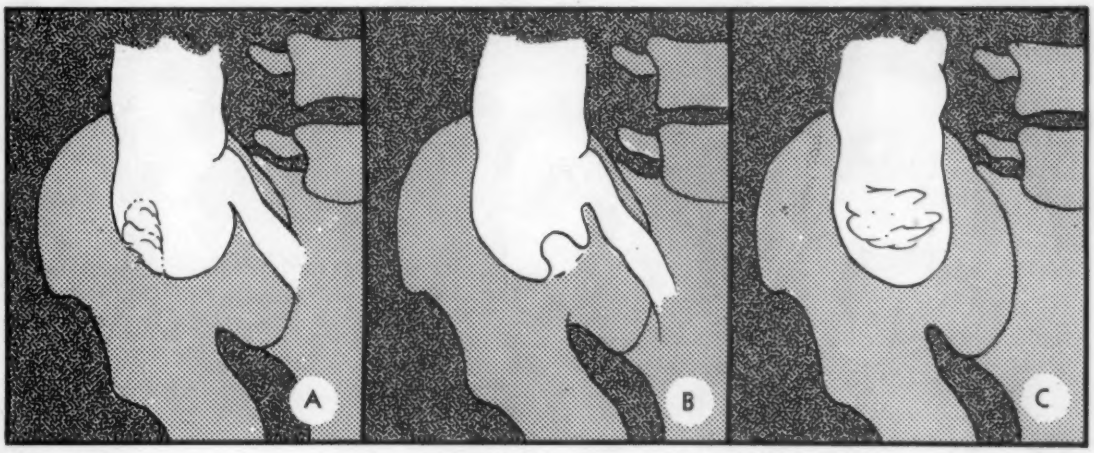

Fig. 6. *Filling defect.* (*A*) Contour irregular; peripheral. (*B*) Contour smooth near base of appendix. (*C*) Central type irregular contour near valve region with ileum not filled.
Benign neoplasms of cecum: polyps, carcinoid, lipoma, fibroma.
Malignant neoplasms of cecum.
Granulomas of cecum.
Appendiceal disease including appendiceal stump.
Intussusception with or without mass (*C* only).

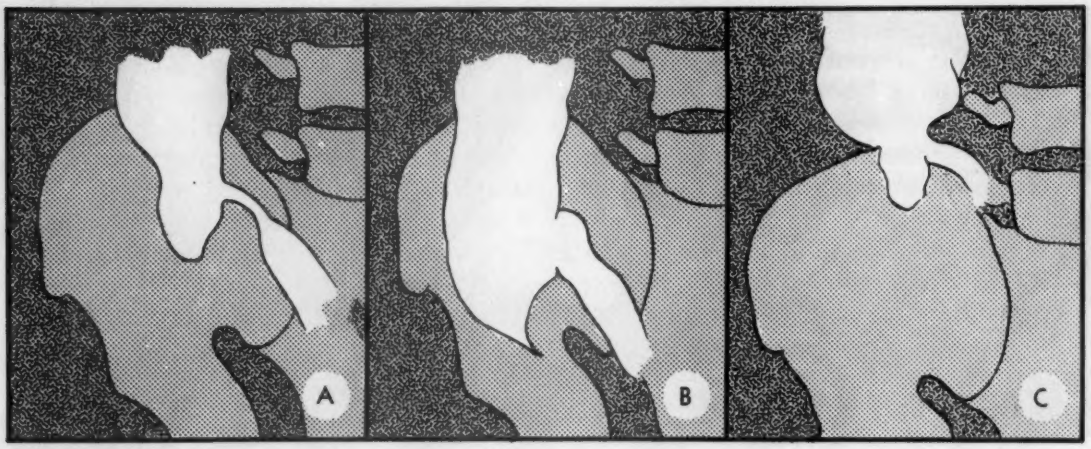

FIG. 7. *Narrowing.* (*A*) Shortening; smooth edges; ileum involved. (*B*) Smooth; conical cecal tip; ileum not involved. (*C*) Shortening; irregular; ileum not involved.
Chronic inflammatory and granulomatous disease: amebiasis, tuberculosis, regional enterocolitis.
Malignant lesions: carcinoma.

pericecal lesion rules out the appendix as a cause for the cecal finding. On the other hand, nonfilling of the appendix in the presence of a cecal or pericecal lesion may or may not be related, and appendiceal disease cannot be excluded.

Mass lesions of the appendix,[44] including abscess, carcinoid, mucocele and carcinoma,[19] may affect the adjacent cecal wall, either as a filling defect or as varying degrees of localized displacement or indentation of the wall (Fig. 5 and 11). The visible effect on the cecum does not necessarily indicate the size of the appendiceal lesion. Mass appendiceal lesions may also cause extrinsic pressure on the terminal ileum. Gross carcinoid lesions of the appendix are relatively rare and overwhelmingly benign.[43] Very rarely does appendiceal carcinoid encroach on the cecum.

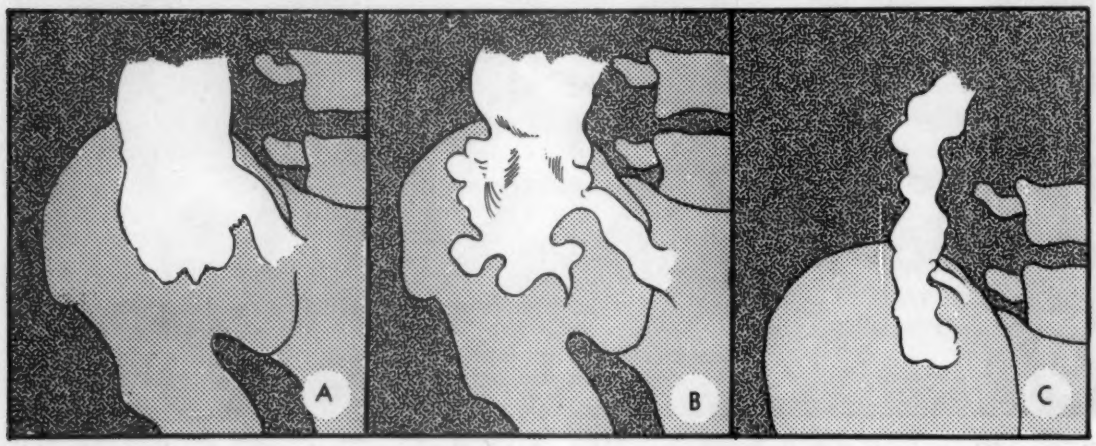

FIG. 8. *Irregular cecal contour.* (*A*) Fine serrated edge; primarily cecal tip; ileum not involved. (*B*) Slight involvement of ascending colon; ileum not involved. (*C*) Narrowing; marked involvement of ascending colon; ileum involved.
Malignancy: carcinoma or lymphosarcoma.
Chronic inflammatory and granulomatous diseases: ulcerative colitis, regional enterocolitis, segmental colitis, tuberculosis, amebiasis, diverticulitis.

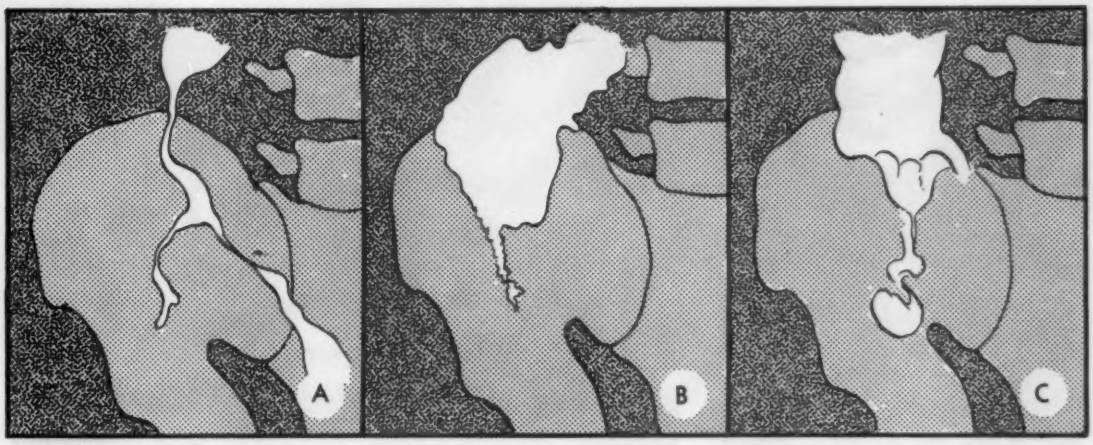

Fig. 9. *Marked irregular narrowing.* (*A*) Fistula formation; fixation; ileum extremely involved. (*B*) Marked serrated edge; ileum not filled. (*C*) Abrupt transition with shelf formation.
Advanced chronic inflammatory and granulomatous disease: tuberculosis, regional enterocolitis, ameboma.
Malignant lesion.
Appendiceal disease.

Mucocele of the appendix[45] is also rare (Fig. 11*A*). Calcific deposits in the mucocele are not infrequent and may be a differential point from other right lower quadrant lesions. Rarely is mucocele of the appendix malignant and in some of these malignant cases, it may rupture and implant on the cecal or ileal wall, leaving the overlying mucosa intact and giving the appearance of an extramucosal intramural lesion. The rare appendiceal carcinoma (Fig. 11*B* and 12*B*) involves the contiguous cecum by pressure or invasion of the walls or lumen.

Invagination of the appendiceal stump[42] following appendectomy may produce a

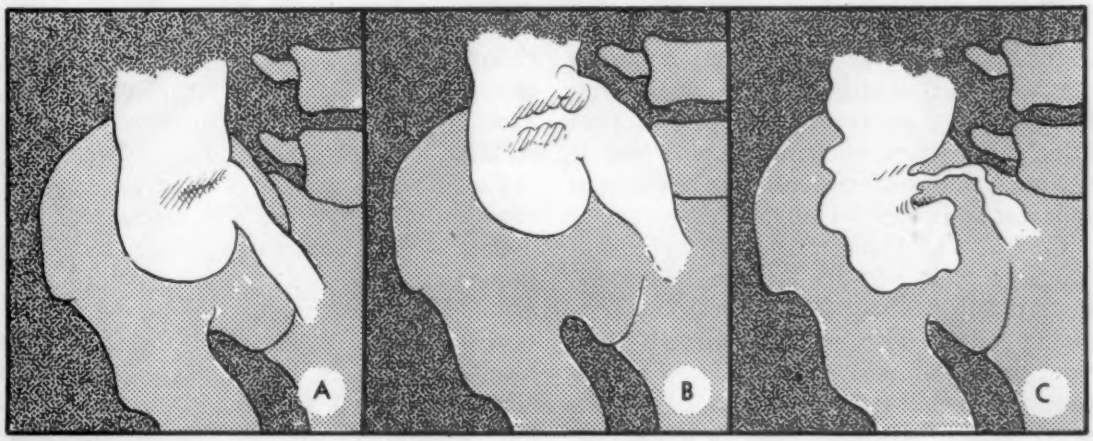

Fig. 10. *Ileocecal region and valve.* (*A*) Ill-defined margins. (*B*) Lobulated margins; ileum involved. (*C*) Irregular margins; body of cecum and ileum involved.
Prominent valve: normal, hypertrophy, edema, prolapse of ileal mucosa.
Benign tumors.
Malignant tumors.
Granulomas and chronic inflammation.
Intussusception: minimal stage, or incompletely reduced.

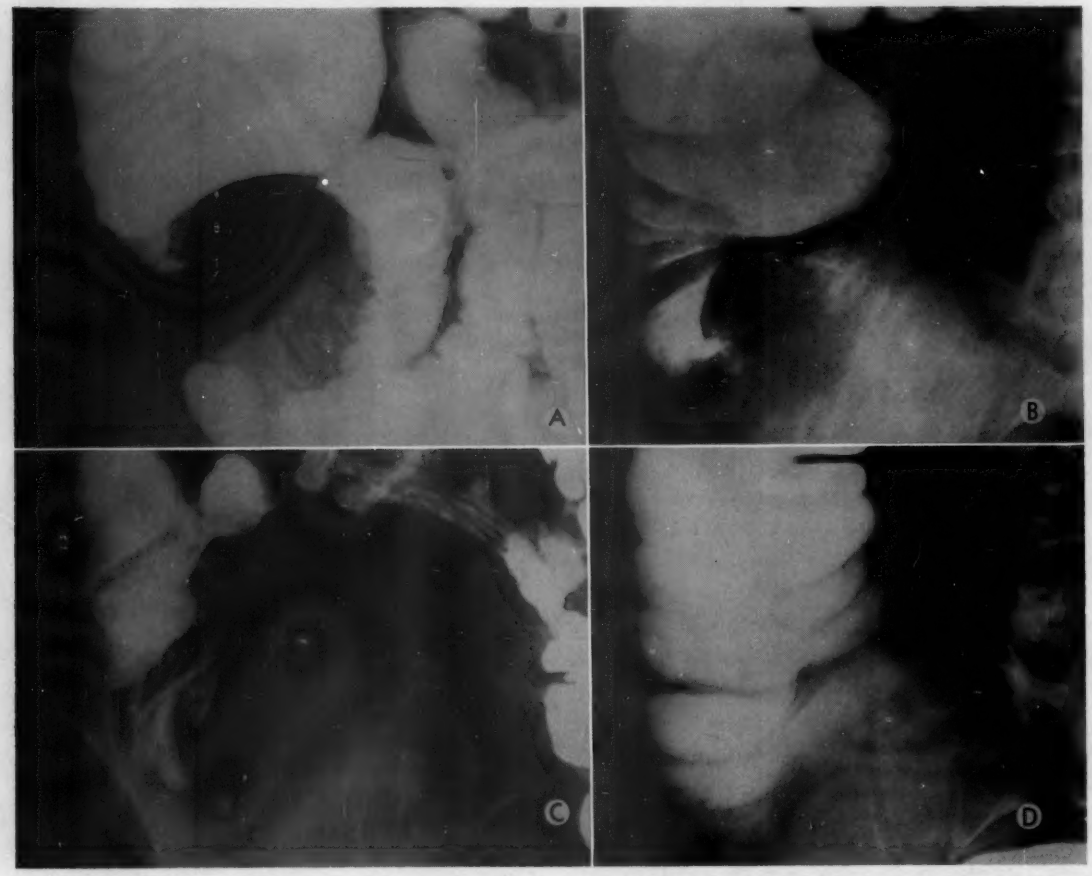

FIG. 11. *Local wall displacement or filling defect.* (*A*) Pathologic diagnosis: mucocele of appendix. (*B*) Pathologic diagnosis: carcinoma of the appendix. (*C*) Operative findings: appendiceal abscess with displacement of the ileum. (*D*) Pathologic diagnosis: carcinoma of cecal tip.

rounded nodular protuberance simulating a tumor, particularly with a double contrast air-barium study. It appears that after some years, the protuberant nodule becomes flattened out to such an extent that it is no longer demonstrable.

Chronic inflammatory masses or *granulomas* (Fig. 5 and 6) include ameboma, hyperplastic tuberculosis, actinomycosis, diverticulitis[22] with granulomatous formation, benign ulcer[9] with granuloma formation, Crohn's disease[12] (limited to the cecum) and other nonspecific granulomas. These granulomas present roentgen findings which closely simulate those of carcinoma and a differential diagnosis is often impossible. The presence or absence of fistulous tract formation with the above lesions does not aid in differentiation although they are more likely to occur with granulomatous than with malignant diseases, except in cases of appendicitis associated with carcinoma.

Radke[33] reported on a large series (119 cases) of ameboma of the intestine. The diagnosis was based on localized tumor-like lesions in patients from whom *Endamoeba histolytica* was isolated or in whom the reaction to complement fixation tests for amebiasis was positive.[3] Roentgen findings were most often interpreted as malignant. Amebic granuloma, when extensive, often shows poor response to treatment because of size and dense scar formation. The presence of *Endamoeba histolytica* in the stool does not always make a definite or a sole diagnosis of amebiasis, since amebic granuloma and carcinoma may coexist. The

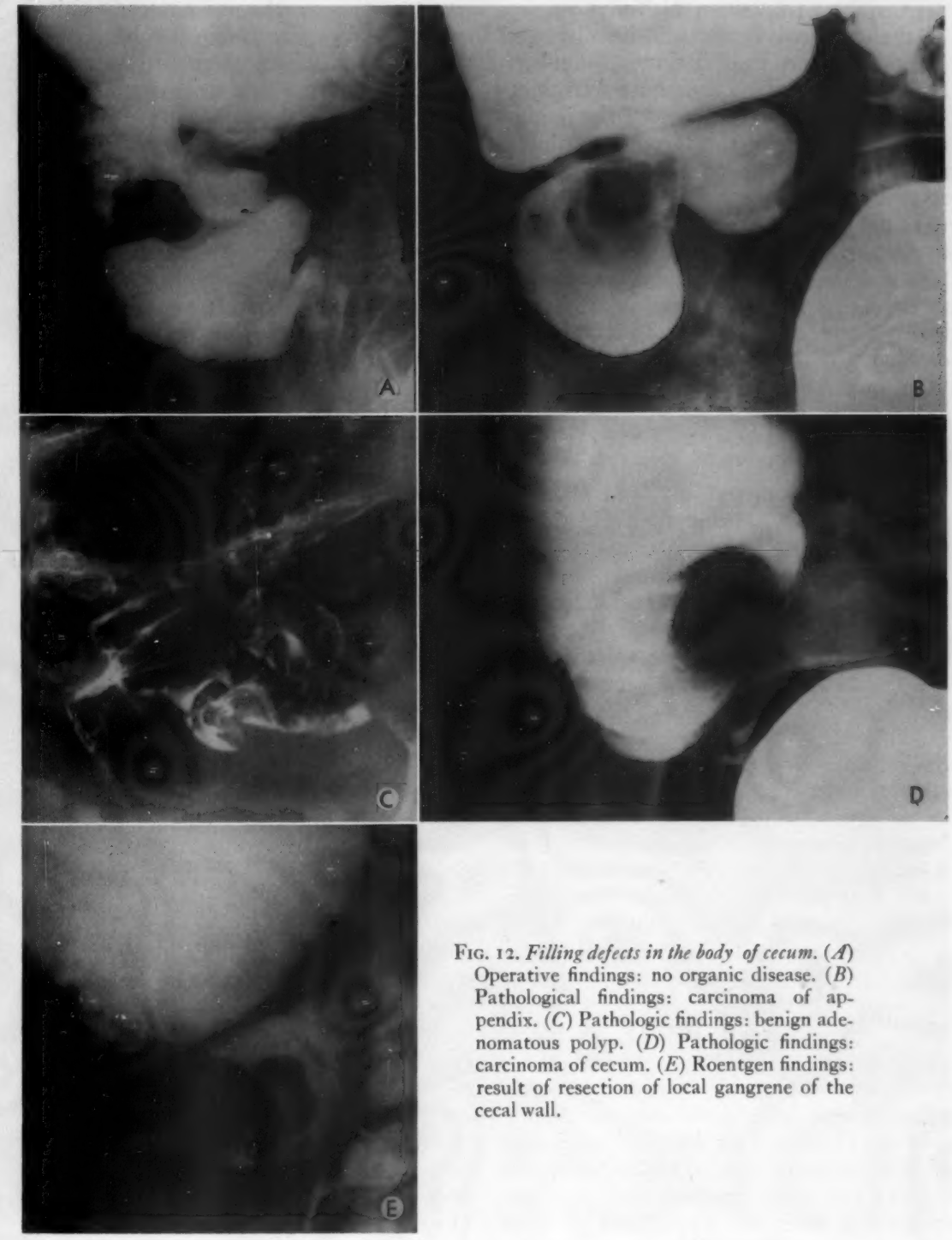

Fig. 12. *Filling defects in the body of cecum.* (*A*) Operative findings: no organic disease. (*B*) Pathological findings: carcinoma of appendix. (*C*) Pathologic findings: benign adenomatous polyp. (*D*) Pathologic findings: carcinoma of cecum. (*E*) Roentgen findings: result of resection of local gangrene of the cecal wall.

lesion may show prompt but temporary response to antiamebic therapy, thus giving the examiner a false sense of security. The carcinoma may appear not unlike residual scarring from an amebic granuloma.

Extensive filling defects and marked

narrowing of the cecum with involvement of the terminal ileum will be discussed together, since the distinction between these roentgen signs is frequently subjective and arbitrary (Fig. 9). Of the inflammatory lesions in this group, amebiasis rarely involves the terminal ileum; nonspecific ulcerative colitis may involve the terminal ileum but this is not common; tuberculosis involves both the cecum and terminal ileum in most cases; nonspecific regional enteritis of the terminal ileum also involves the cecum in a small percentage of cases (Fig. 7).

In an excellent presentation of the subject, Golden and Ducharme[18] discuss the significance of deformity of the cecum in amebiasis without the formation of ameboma. The cecum was involved in about one-third of the cases studied whether or not clinical symptoms were present. Ninety per cent of the cases had *Endamoeba histolytica* in their stools. The cecal deformity varied considerably: in some cases it was slight, amounting only to a narrowing of the tip; in a few cases, the cecum was so shrunken that its shadow measured only 2–3 cm. in diameter. In the great majority the outline was smooth, but in some the cecum was irregular in outline and narrow, without much shortening. The size and outline of the cecum often changed a little from time to time; this was particularly noticeable when the deformity was slight. Localized tenderness was usually elicited on fluoroscopic examination. In none of the cases was the terminal ileum narrowed or intrinsically deformed. Undoubtedly, some of the cecal deformity was due to spasm, since relaxation or decreased deformity occurred with antiamebic treatment. No palpable masses were associated with their findings. Despite their detailed description of the roentgen findings, Golden and Ducharme are quick to point out that the question of amebiasis is suggested by them and not made as a specific diagnosis.

Ulcerative colitis or *segmental colitis* which, at the time of the examination, may be limited to certain segments of the large intestine, such as the cecum, may subsequently show progressive involvement of more distally placed segments, although the reverse findings are more common. The incidence of involvement of the ileum in chronic ulcerative colitis, as reported in the literature, varies from 1–40 per cent. Ileal involvement may occur early in the disease. In most cases the extensive involvement of the colon with concurrent involvement of the ileum leaves little doubt as to the correct diagnosis of chronic ulcerative colitis.[24] There are certain lesions, however, which involve simultaneously the colon and small intestine in a nonspecific inflammatory process. These cases, depending on the site of the lesion and whether or not they are stenotic, Finkelstein[15] divides into three major types: (*a*) right sided colitis or diffuse colitis with continuous involvement of a stenotic terminal ileitis; (*b*) right sided colitis associated with a dilated, stiff terminal ileum; and (*c*) regional or segmental colitis associated, but not continuous with, regional enteritis. Complications which occur with colitis are: obstruction, perforation, fistula formation, adhesions and rarely, malignancy. Some authors feel that regional enteritis,[40] ileocolitis[27] and regional colitis[26] are manifestations of the same disease in different segments of the intestine, but consider ulcerative colitis a different entity because it first involves the sigmoid and rectum in 95 per cent of the patients.

Tuberculosis of the intestinal tract was studied by Brown and Samson[6] (as reported by Golden), who in 184 cases of pulmonary tuberculosis with intestinal lesions found that the disease was present in the small intestine alone in 6 per cent, in the cecum alone in 19 per cent and in both places in 75 per cent of the cases. Practically all cases with intestinal involvement have active or old pulmonary tuberculosis. The ulcerative type is much more common than the hyperplastic type. Alteration or absence of the mucosal pattern, irregularity of margins of the barium shadow, hyperirritability or marked spasm are fairly

typical of the ulcerative type of tuberculous lesions. Marked spasm of the cecum, so-called "Stierlin's sign," is a very common occurrence in ileocecal tuberculosis, but Schinz *et al.*[37] state that "Stierlin's sign" is in no wise pathognomonic of tuberculosis since it is also observed commonly in every other form of inflammatory alteration involving the ileocecal region. In various stages of ileocecal tuberculosis, there are cases where roentgen differentiation from ulcerative colitis, amebiasis or other granulomas is impossible. Differentiation from carcinoma may also be impossible since carcinoma can extend into the ileocecal region or into the ascending colon (Fig. 14).

The ileocecal valve (Fig. 13) often requires special studies to be demonstrated roentgenographically. In barium enema examinations most normal cases show regurgitation into the terminal ileum. Nonregurgitation, however, does not imply a pathologic state. If deemed necessary, administration of oral contrast meal may be used to study the terminal ileum or ileocecal valve. There are variations in position, shape and thickness of the valve area so that no exact normal dimensions or configuration can be given.[16] At times the valve region appears

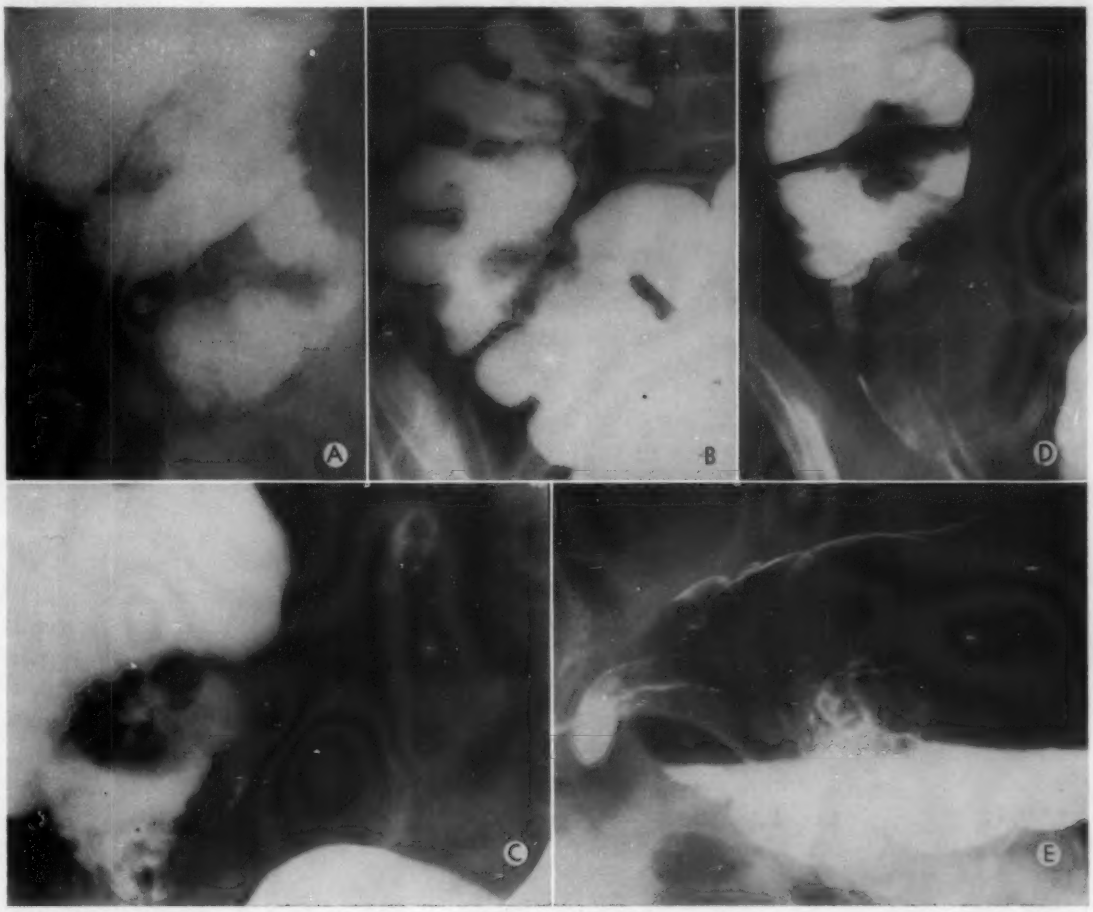

Fig. 13. *Prominent or enlarged ileocecal valve region.* (*A*) Operative findings: no pathologic process found in region of valve; several polyps in ascending and transverse colon. (*B*) Operative findings: edema of valve. (*C*) Pathologic diagnosis: submucous lipoma of valve. (*D*) Pathologic diagnosis: carcinoma of ileocecal valve. (*E*) Lateral decubitus double contrast study of case *D*, showing the lesion in the valve area to better advantage.

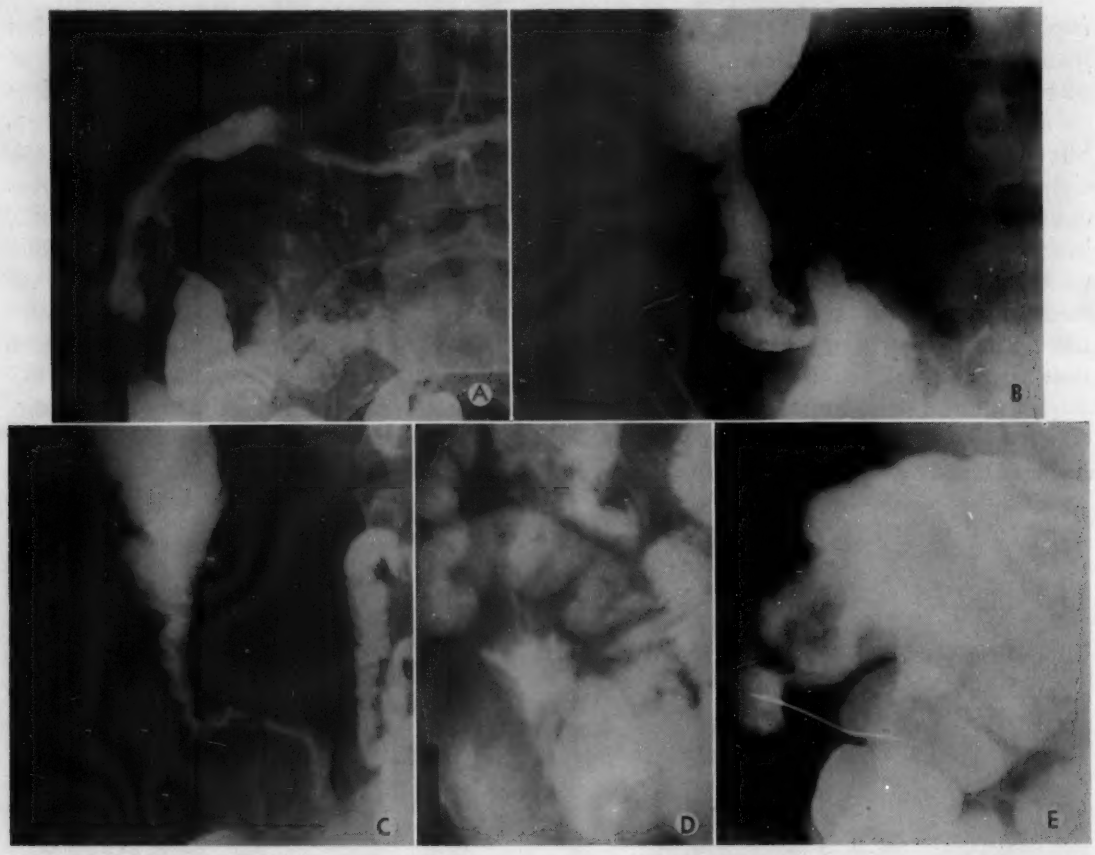

FIG. 14. *Extensive irregular narrowing of the cecum with involvement of adjacent segments.* (*A*) Pathologic diagnosis: extensive carcinoma of cecum, ascending and transverse colon, and terminal portion of ileum; no evidence of ulcerative colitis. (*B*) Pathologic diagnosis: ileocecal tuberculosis. (Courtesy of Dr. John A. Evans, New York Hospital, New York, New York.) (*C*) Pathologic diagnosis: ileocolitis with fistula formation at cecal tip. (Courtesy of Drs. C. Foustanos and M. Georges, New York, New York.) (*D*) Pathologic diagnosis: amebiasis of cecum, ascending colon with perforation and abscess formation. (*E*) Pathologic diagnosis: carcinoma of cecum and ascending colon.

prominent though at operation it shows no organic lesion. The valve may be indirectly affected by some adjacent lesions—especially those about the ileocecal region, such as retrocecal or pericecal adhesions or masses—causing it to protrude or appear enlarged. Prolapse of the ileal mucosa through the valve or edema of the lips of the valve may occur, but they are rare. Hypertrophy is another cause of prominent ileocecal valve lips. The most important cause however is tumors, especially those which are malignant. Tumor formations, while still small, are likely to quickly interfere with the function of the valve and therefore give rise to early symptoms; however, since the lesions are small, they are very difficult to demonstrate. As Hinkel[20] stated, when an unusual or asymmetric filling defect is present in the ileocecal region which cannot be proved to be either ileal mucosa, or the upper or lower lip, surgery is indicated.

Intussusception in the ileocecal region is not difficult to identify by barium enema examination, with the rather typical concertina configuration of the filling defect. In infants and children, no organic cause may be found although such lesions as benign polyps, Meckel's diverticula, or foci of ectopic pancreatic tissue may be initiating factors in a small percentage of cases. In

FIG. 15. *Irregular narrowing and shortening of the cecum.* (*A*) Operative findings: no organic pathology. (No evidence of amebiasis or other infectious process revealed by complete clinical and laboratory workup.) (*B*) Pathologic diagnosis: carcinoma of cecum. (*C*) Operative findings: pericecal inflammatory disease. (*D*) Pathologic diagnosis: carcinoma of cecum. (*E*) Operative findings: granulomatous pericecal mass secondary to appendicitis. (*F*) Clinical diagnosis: chronic ulcerative colitis.

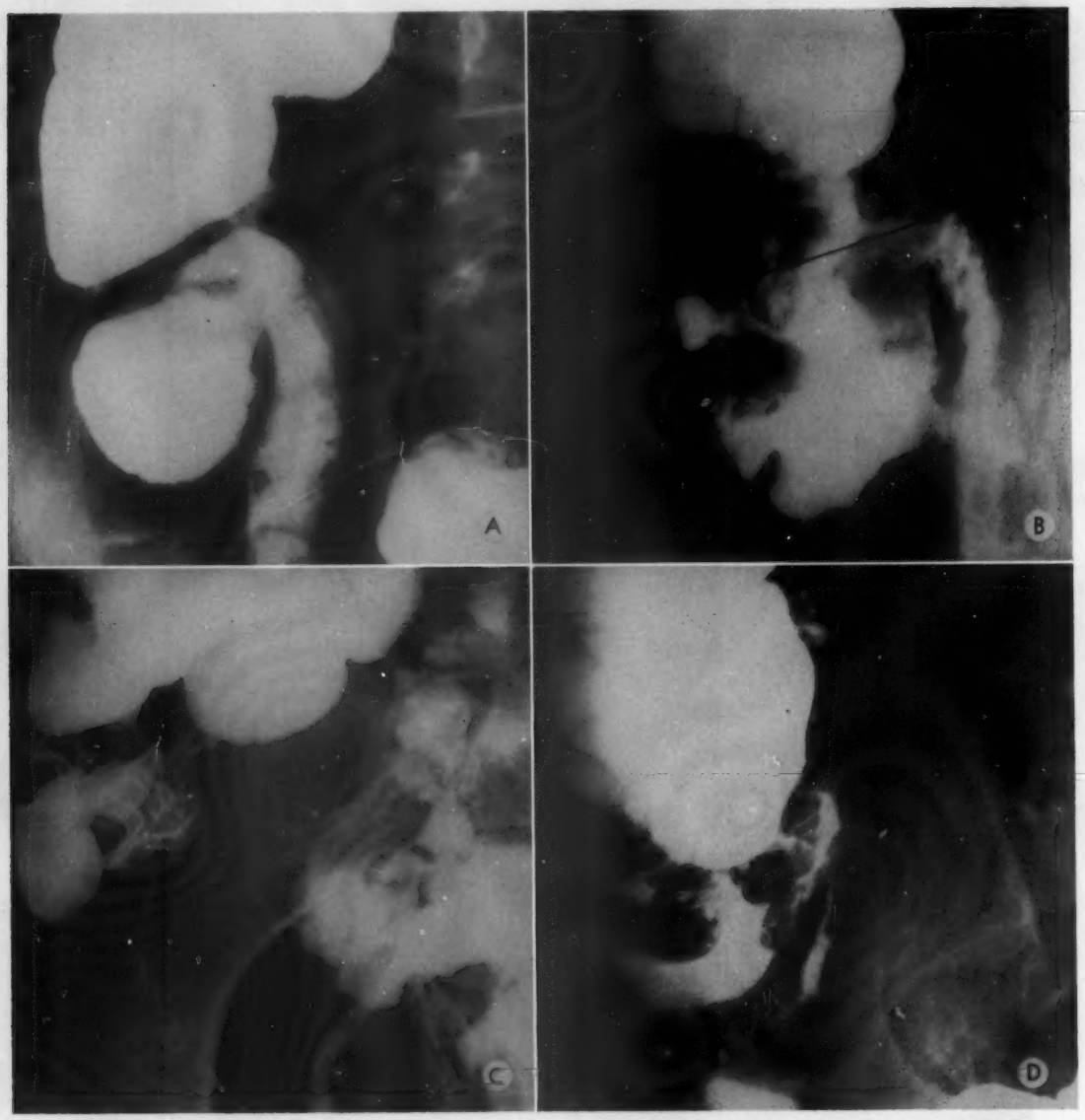

FIG. 16. *Irregular contour.* (*A*) Roentgen findings: fine serrated contour of cecum; ileum apparently not involved. Clinical diagnosis: ulcerative colitis. (*B*) Pathologic diagnosis: carcinoma of the cecum with involvement of the ileocecal valve. (*C*) Roentgen findings: irregular and contracted cecum; extensive involvement of the ileum. Pathologic diagnosis: regional enterocolitis. (*D*) Roentgen findings: irregular contour, appendix filled. Clinical diagnosis: diverticulitis.

adults, intussusception in the ileocecal region is relatively rare but in the great majority of cases it is the result of an organic lesion present in the region. Therefore, the mere diagnosis of intussusception is not sufficient and the underlying lesion which is frequently obscured may be either benign or malignant.

DISCUSSION

The spectrum of each individual disease process is broad, extending from the very minimal deviation from the normal to the maximum degree of development. This applies to the clinical, pathologic, laboratory and roentgen manifestations and frequently each is independent of the other so

that strict correlation rarely exists. In inflammatory lesions, the clinical manifestations may far outrun the roentgen aspects so that roentgenologic studies are instituted fairly early in the development of the process. Roentgen findings, therefore, may be observed which are equivocal or very minimal, but the examiner's attention is focused on them because of the prominent clinical symptoms, *e.g.*, amebiasis[21] or segmental colitis. With advance of the inflammatory process, the clinical picture and the laboratory findings become so obvious that they leave little question as to the interpretation of the roentgen findings, as in the case of ulcerative colitis, ileocecal tuberculosis or regional enteritis with cecal involvement.

If, on the other hand, the clinical symptoms lag far behind the roentgen manifestations, it is much more likely that the roentgenologic study will be undertaken at a time when the lesion and its roentgen signs are in an advanced stage so that the problem of minimal findings no longer has to be considered, as occurs in a large carcinoma of the body of the cecum. When the clinical manifestations force the patient to see his physician, in the majority of cases of carcinoma, the roentgen findings are already far advanced so that the differential diagnosis between early inflammatory lesions and carcinomas need not even be considered. No one questions that there must exist, asymptomatically or "silently," a phase when the carcinoma must be present and could reveal minimal roentgen findings. Unfortunately, either the roentgen method is too crude to detect these early changes or else a chance roentgen study at this time does not lead to the exhaustive study of the cecal region since the clinical manifestations do not point in that direction. Furthermore, despite exhaustive study of the cecal region, it is surprising how few unexpected lesions, such as polyps, for instance, are found in the cecum. This is not to imply that carcinoma of the cecal body must originate as a silent polyp, but it does point out the difficulty in discovering a "silent" lesion in the cecum such as a polyp of 1 cm. in diameter, where so frequently a much larger lesion may be obscured or missed in the distended blind pouch. It should also be apparent that an inflammatory granulomatous process, such as an ameboma or a hyperplastic tuberculous granuloma, which grows to a large size with minimal or indefinite clinical manifestations, would have the roentgen aspects indistinguishable from a carcinoma.

On the other hand, a lesion affecting primarily the ileocecal valve junction, which early in its evolutionary development may quickly lead to overt clinical manifestations, will more likely direct immediate and thorough study to this area and therefore uncover small lesions of all types, including carcinoma (Fig. 13, *D* and *E*).

The correct interpretation of the roentgen findings as depicted at any particular point in the spectrum of a disease process depends not only on the acumen of the radiologist and the proper use of his methods, but also to a very great extent on the clinical and laboratory manifestations which should focus the examiner's attention in the right direction. In other words, there must be proper, but not premature or forced, correlation of the roentgen signs with all available clinical and laboratory data.

A thorough examination of the intestine is obviously essential. An interpretation of the findings made in retrospect is also of value in accumulating data for scientific studies, as well as for the case at hand, to help determine the rate of progression or regression of the disease process. It must be stressed, however, that there are inherent limitations in the roentgen method of examination which cannot be entirely eliminated, either by stressing meticulousness or by practicing retrospection.

SUMMARY

The major cecal and paracecal lesions have been presented in terms of their descriptive roentgen findings or signs. The differential diagnosis of these lesions has

been discussed with the aid of diagrammatic sketches and roentgenographic reproductions.

The illustrations clearly show the concept of convergence where dissimilar pathologic lesions display similar roentgen signs; and the concept of divergence where similar pathologic lesions display dissimilar roentgen signs.

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PLAIN ROENTGENOGRAPHY IN DIAGNOSIS OF CHRONIC ULCERATIVE COLITIS AND TERMINAL ILEITIS*

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THE concept that the fulminating form of ulcerative colitis is diagnosable from the plain roentgenogram has been set forth very ably and convincingly in a recent article by McConnell, Hanelin, and Robbins.[4] That other forms of advanced chronic ulcerative colitis can be recognized on plain roentgenograms was not mentioned in this paper, in Frimann-Dahl's[2] monograph on the acute abdomen, or elsewhere in the available literature. We have been able without prior clinical information to suggest the diagnosis of chronic nonfulminating ulcerative colitis in a few cases from abdominal roentgenograms in which the colon was not opacified by contrast material, but was outlined by retained gas. On one occasion terminal ileitis was recognized under similar circumstances. We have been able to find no reference to any other case of regional enteritis being diagnosed in this fashion.

The diagnosis of these conditions from the plain roentgenogram is of some value, beyond the satisfaction of making a diagnosis by the most economical and fundamental means at hand. If either colitis or enteritis is suggested, one may be inclined to use less drastic methods of preparing the bowel and more caution in performing the examination.

We have collected from the hospital records of the past five years all well authenticated cases of ulcerative colitis which had some roentgen examination of the abdomen not associated with contrast filling of the colon. These examinations include studies of the gallbladder, upper gastrointestinal tract, small bowel, and urinary system, as well as the usual plain roentgenograms. Many more cases of ulcerative colitis have been seen during this period, but in a relatively small number were plain roentgenograms or other suitable examinations obtained to permit study of this particular roentgen aspect of the disease.

There were 10 cases of late stage chronic colonic disease under which we have included 3 classifiable as the fulminating ulcerative type: one obstructed case, one which we presume to be a burned-out case of ulcerative colitis, and one in which the alterations are likely due to chronic catharsis. By late stage is meant the shortened, pipe-like colon, usually thick-walled, and with practically complete loss of haustral pattern. Of these, 9 could easily be recognized in retrospect and we believe should be identified as easily in prospect, provided that the observer carefully inspects the character of the intestinal gas shadows. The illustrations presented should support this contention.

Allowing for the fact that the bowel on plain roentgenograms is delineated by the fortuitous retention of negative contrast material (gas) rather than the deliberate introduction of positive contrast by the barium enema, the roentgen alterations in the two types of examination generally correspond and conform to the pathology. The colon appears tubular due to the absence of haustration. It may vary in caliber from a moderate decrease to a moderate increase compared to the normal, excluding the extreme dilatations seen in the fulminating or exceptional cases. Frequently, thickening of the bowel wall may be perceived. In none of our cases was gaseous delineation of ulcers visible. The nodular mucosal thickening mentioned by McConnell *et al.*[4] in their fulminating cases was

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seen only in our similar cases.

We were able to find only 2 analyzable early (acute) cases of ulcerative colitis. In these the pathologic process had not progressed beyond the stage of ulceration, and apparently the resiliency and flexibility of the wall had not yet been appreciably compromised. As expected, in neither case could the diagnosis be suspected from the plain roentgenograms.

REPORT OF CASES

Case I (Fig. 1, *A* and *B*). Chronic nonfulminating ulcerative colitis. A fifty year old white female known to have ulcerative colitis for fifteen years was admitted with complaints of progressively severe diarrhea, melena, and abdominal cramps. Subsequently she had a total colectomy.

Case II (Fig. 2, *A* and *B*). Chronic nonfulminating ulcerative colitis. A twenty-five year old white female known to have ulcerative colitis for two years was admitted on May 21, 1958 with complaints of persistent and increasing abdominal pain for several days and no bowel movement for two days. A roentgenogram (not shown) demonstrated intestinal obstruction and laparotomy established the presence of a transverse colonic lesion. A cecostomy was performed. When her condition was favorable, a transverse colectomy was done. Pathologic diagnosis was anaplastic carcinoma in chronic ulcerative colitis.

The patient improved and was discharged. She was re-admitted on September 13, 1958 with fever, headache, wound drainage, diarrhea, and abdominal pain. She responded well to revision of the colostomy and conservative management (Fig. 2, *A* and *B*).

Case III (Fig. 3). Exacerbation of chronic ulcerative colitis. A thirty-one year old white female had symptoms of fourteen months' duration; however, an increase in severity was noted the last five months. The complaints included liquid stools with blood and mucus, abdominal

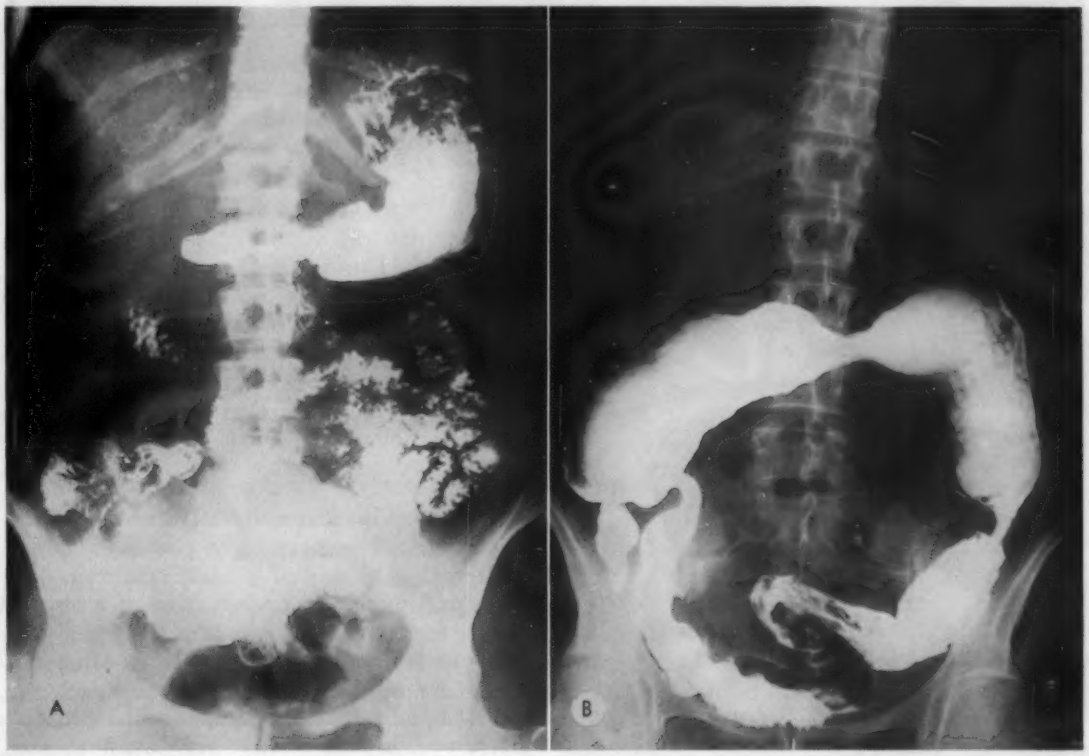

Fig. 1. Case I. (*A*) Small bowel series. The hepatic flexure and transverse colon are most easily visualized and display a tubular appearance due to absent haustration. Thickening of the bowel wall is evident. (*B*) Confirmatory barium enema roentgenogram.

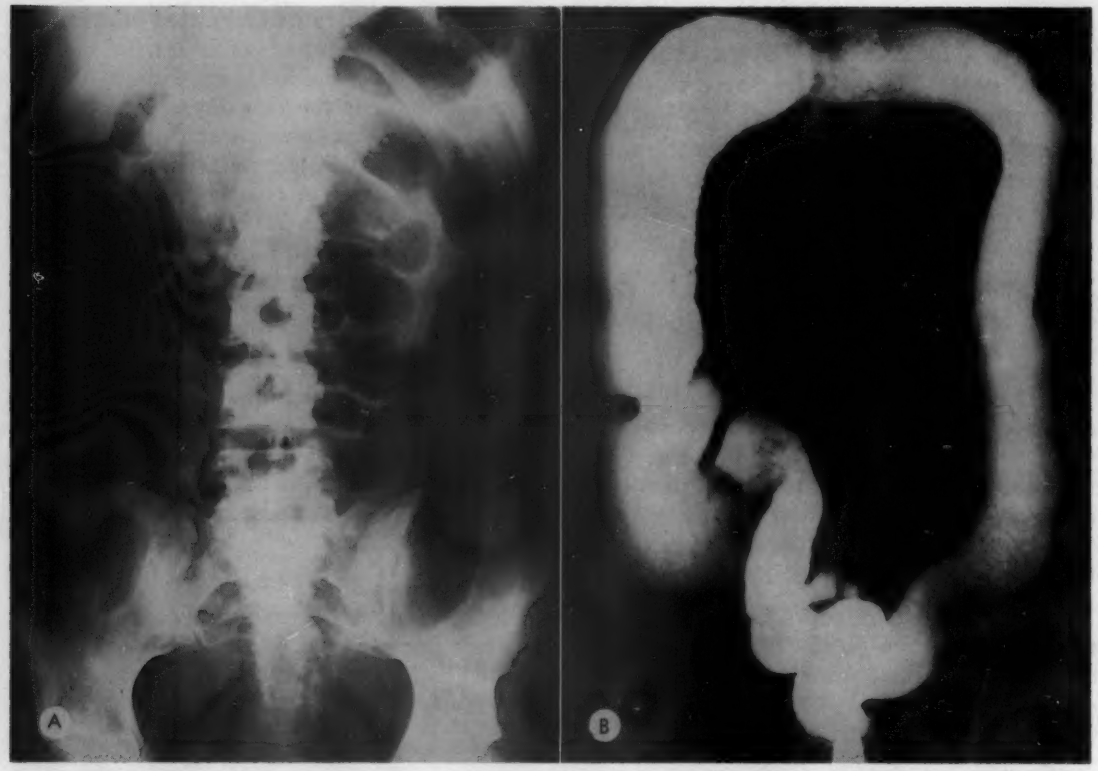

FIG. 2. Case II. The colonic shortening is partially due to previous transverse colectomy. (*A*) Plain roentgenogram. The alterations are beautifully exhibited in the descending colon. The thickening of the wall is quite apparent. The cecum also appears to be abnormal. The transverse colon is obscured by the distended small bowel. (*B*) The barium enema roentgenogram reveals obvious ulceration.

FIG. 3. Case III. Only plain roentgenograms were obtained in this case. The transverse colon is clearly diseased. A similarly abnormal gas-filled viscus above the full bladder is probably the sigmoid.

cramps, nausea, vomiting, and a 50 pound weight loss. During this time she had received 20 pints of blood in a ten week hospitalization elsewhere. After failure of medical management she had a subtotal colectomy and ileostomy, but a continuance of symptoms necessitated an abdominoperineal resection after which she did well. Pathology reported typical ulcerative colitis.

CASE IV (Fig. 4, *A* and *B*). Burned-out ulcerative colitis. This sixty-seven year old white female with a history of nausea, vomiting, diarrhea, and upper abdominal pain of a chronic nature for many years was admitted chiefly because of extreme weakness and loss of energy. Sigmoidoscopy was essentially negative. She

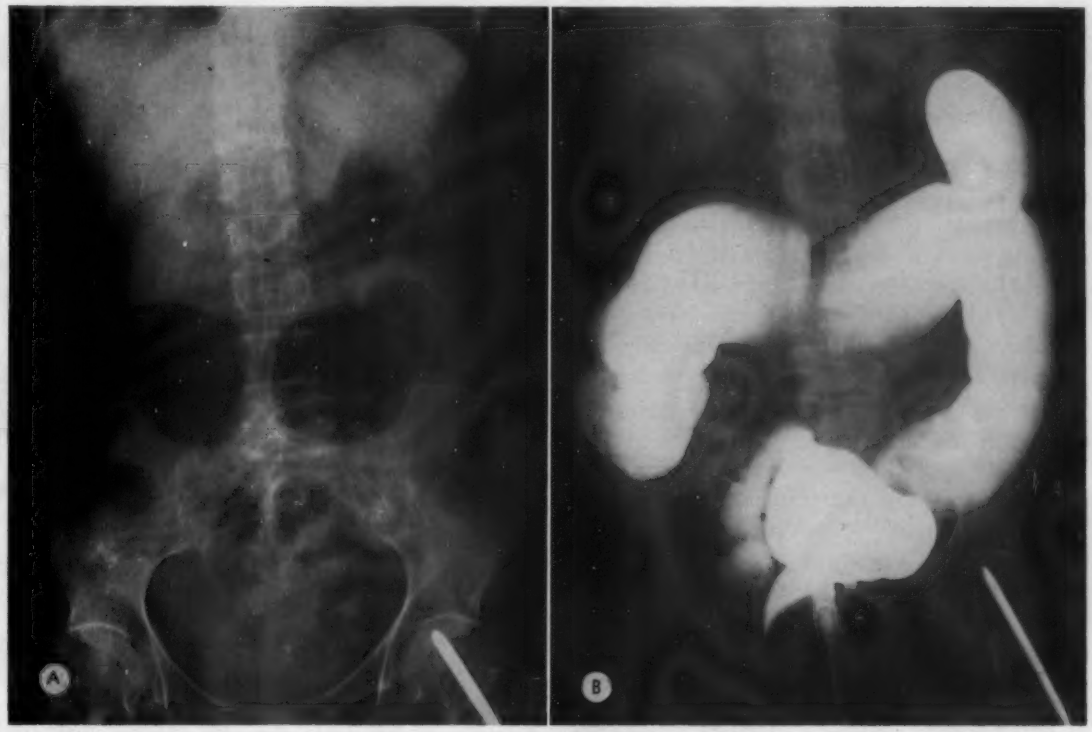

Fig. 4. Case IV. (*A*) Gas is trapped in patternless and dilated transverse colon due to cicatricial stenoses near the hepatic and splenic flexures which were visualized during the barium enema study (*B*), but are not well demonstrated in the frontal projection. This was an inactive or burned-out case. Despite transverse colonic distention, smooth contour of wall and absence of small bowel distention differentiated this from fulminating colitis.

improved considerably on a supportive medical regimen.

Comment. This was thought to be an inactive or burned-out phase of chronic ulcerative colitis. The amount of distention in the transverse colon brings to mind that often seen in fulminating ulcerative colitis. However, the contour of the gas-outlined colonic wall was smooth due to the absence of mucosal pseudopolypoid hypertrophy. In addition, and most important in our opinion, was the absence of small bowel ileus which was consistently present in McConnell and co-workers' cases and occurred in all of our fulminating cases as well.

Case V (Fig. 5, *A* and *B*). Fulminating ulcerative colitis. An eighty year old white male with intermittent diarrhea for nine years was admitted with increasingly severe cramps and diarrhea, anorexia, and weight loss. Medical therapy and Wangensteen intubation were of no avail and his condition deteriorated progressively until his death, forty-one days after admission. Autopsy revealed chronic ulcerative colitis and ileitis, ascites, gastric ulcers (probably terminal), and distention of intestines, gallbladder, and urinary bladder.

Comment. Both the plain roentgenograms and the clinical course were such that this case was placed in the fulminating classification. The colon was somewhat larger in caliber than on the barium study one month earlier, but the transverse colon was not appreciably dilated when compared to the normal. It was much less dilated than those in the illustrations appearing in the paper by McConnell *et al.* Furthermore, it was not possible to see nodular mucosal thickenings in the colon. The common denominator for the diagnosis of fulminating colitis aside from the absence of haustra seems to be distention of the small bowel.

Case VI (Fig. 6, *A* and *B*). Fulminating ul-

cerative colitis. A fifty year old white male with ulcerative colitis for twenty years was admitted on February 11, 1958 after several days during which he passed little stool and his abdomen became distended and painful. He had lost considerable weight. The abdomen was also tender, and quiet. Bowel perforation apparently occurred between the time of the plain roentgenogram (February 11, 1958) and the barium enema study (February 13, 1958). The patient died shortly afterward. There was little doubt of the fulminating character of this process. An autopsy was not obtained.

CASE VII (Fig. 7, *A* and *B*). Fulminating ulcerative colitis. A sixty-six year old white female with a long history of ulcerative colitis and recurring attacks of diarrhea, melena, and abdominal cramps had refused surgery on numerous occasions. She also had rheumatoid arthritis, but the immediate reason for admission on August 1, 1957 was a dermatitis thought to be due to drug sensitivity. She did reasonably well on conservative management and was scheduled for discharge when on September 19, 1957, she had a sudden explosive diarrhea which persisted. The abdomen progressively distended. Perforation was suspected and emergency ileostomy was performed on September 26, 1957. At surgery the cecum and ileum were distended and there was a fibrinous peritoneal exudate. Temporary improvement followed but a relapse occurred after a sudden onset of abdominal pain on October 8, 1957 and the patient died two days later.

Autopsy disclosed the colon to be distended and its wall paper-thin in places. The lumen contained large amounts of thick fecal material throughout. Isolated ulcers which did not resemble the typical lesions of chronic idiopathic ulcerative colitis were present throughout. The perforation of the colon measured 10 cm. in diameter and did not appear to be in an ulcer bed. Apparently, secondary to the perforation of the colon were acute peritonitis, a pelvic abscess, and an ileovaginal fistula. In the autopsy report the question of megacolon was raised.

Comment. This was an unusual case, even exclusive of the plain roentgenogram which was highly suggestive of megacolon except

FIG. 5. Case v. Fulminating colitis. (*A*) April 6, 1955. Barium enema study. (*B*) May 5, 1955. Plain roentgenogram. The colon is moderately dilated and its wall is thickened. There is no definite nodular mucosal hyperplasia. Lack of haustration together with dilatation or ileus of other hollow viscera is the main criterion of fulminating colitis.

for the age of the patient. One can only speculate upon the possibility of an underlying primary megacolon. The bizarre appearance of the barium enema was startling. Filling defects in the sponge like colonic cavities were originally thought to be giant inflammatory pseudopolyps, but were revealed at autopsy to be fecal aggregates. Although the retrograde filling of the colon and terminal ileum was accomplished easily and without indication of significant obstruction, barium was retained in the colon for a long time. In a colon so severely and chronically diseased, this fact lends no support to any hypothesis relative to megacolon. The clinical course and roentgen findings are indicative of fulminating disease.

Case VIII (Fig. 8, *A* and *B*). Ulcerative colitis with obstruction. A fifty-six year old white female with a history of ulcerative colitis for thirteen years was admitted during an acute exacerbation of diarrhea with blood, mucus, pus, and severe generalized abdominal pain. The abdomen was distended, quiet, and tender. After obstruction was diagnosed from the scout roentgenogram (Fig. 8 *A*), a transverse colostomy was done. Distention extended to the left transverse colon where there was what appeared to be an inflammatory stenosis. Distal to this, the colon was markedly contracted and appeared typical of ulcerative colitis. The transverse colon also showed chronic disease but the cecum and ascending colon seemed to be uninvolved except for distention. The patient remained febrile until an ileostomy and drainage of an abscess at the colostomy site were performed two months later, after which she improved and was discharged to return subsequently for colectomy.

Case IX (Fig. 9, *A* and *B*). Probable cathartic colon. This fifty year old white female epileptic had constipation, blood stained stools, and a sensation of rectal blockage, but denied diarrhea and mucus. Constipation was becoming more intractable and was accompanied by crampy abdominal pains, weight loss, progressive weakness, chills, fever, and dysuria. A di-

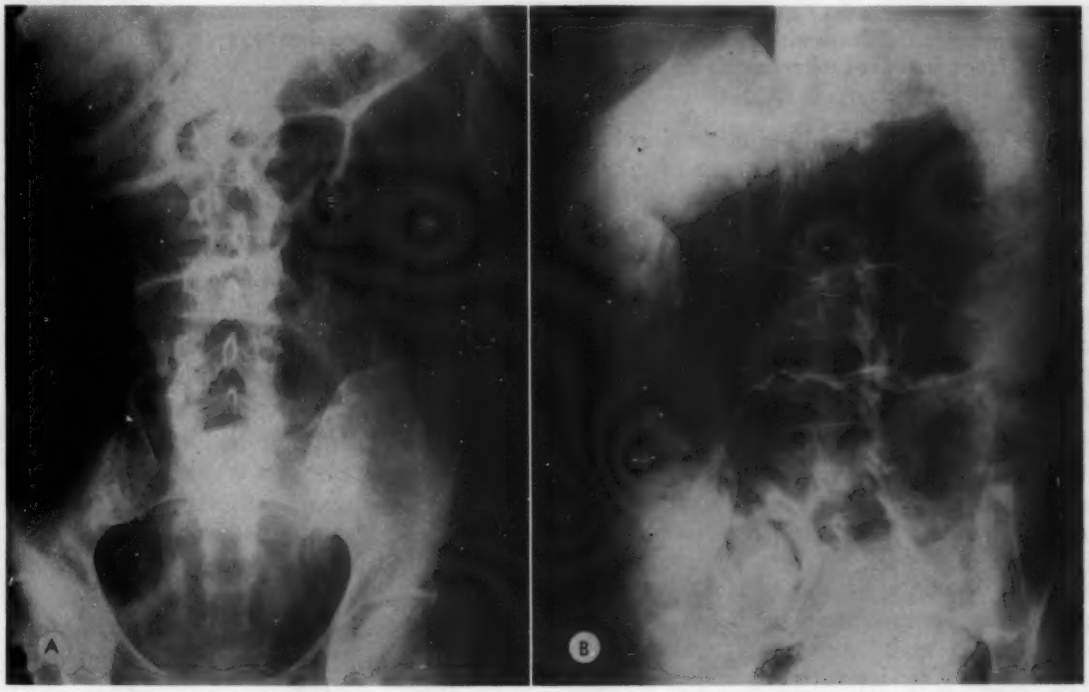

Fig. 6. Case VI. Fulminating colitis. (*A*) February 11, 1958. Plain roentgenogram. Generally distended patternless colon. The nodular mucosal hyperplasia is not definite. There is some increase in the thickness of the colonic wall. No free air is seen. (*B*) February 13, 1958. Barium enema roentgenogram. Extravasation of barium between the dilated small bowel loops proves perforation.

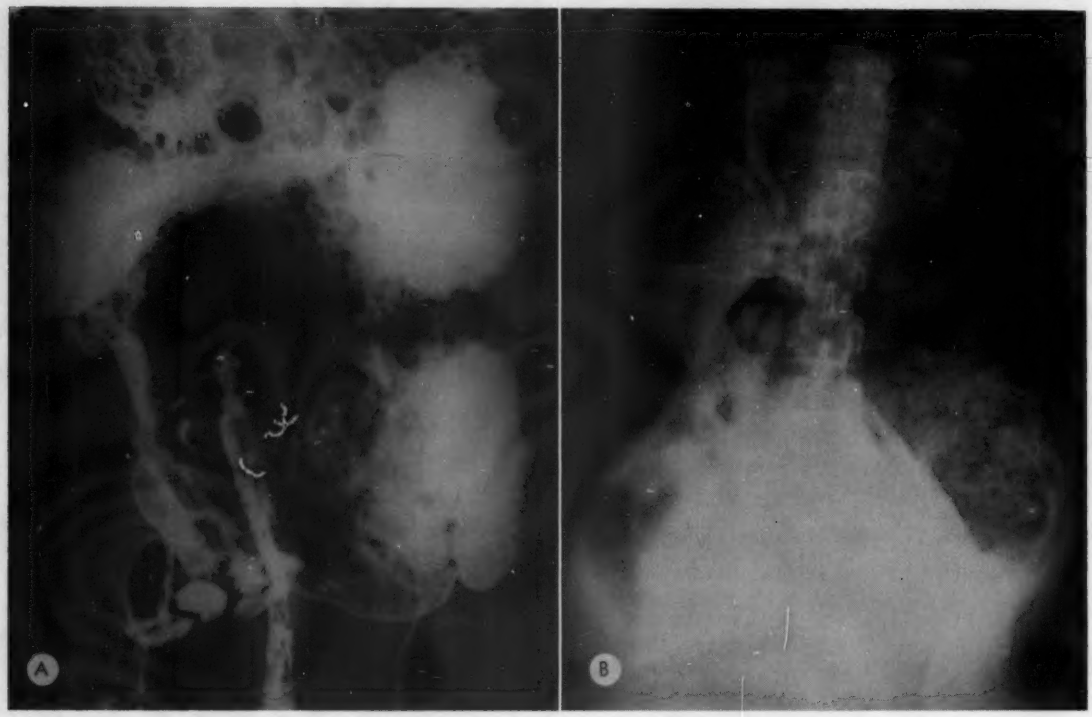

FIG. 7. Case VII. Fulminating colitis. (*A*) August 19, 1957. Barium enema study. Rigid vertical rectum and rectosigmoid with deep ulcerations. Proximal to this, the colon was divided into large easily communicating saccular compartments of sponge-like appearance. The filling defects were fecal masses. Involvement of both appendix and ileum with probable fistulation from latter. (*B*) September 26, 1957. Plain roentgenogram. Huge fecal filled colon resembling megacolon. Pelvic mass.

agnosis of ileocolitis had been made elsewhere. She admitted being a chronic user of milk of magnesia. On sigmoidoscopy the mucosa was observed to be granular and hypertrophic and to bleed easily, but true ulcerations were not seen. The patient remained on cathartics and responded well to a program directed primarily to correction of malnutrition. The diagnostic impression favored cathartic bowel over ulcerative colitis.

CASE X (Fig. 10, *A* and *B*). Terminal ileitis. A previously healthy twenty-four year old white female reported upper abdominal cramps, anorexia, nausea, vomiting, and constipation beginning one week prior to admission. The pain subsequently migrated to the lower abdomen. She appeared thin and dehydrated and was febrile. The abdomen was distended, silent, and generally tender with poorly localized rebound. The diagnosis of terminal ileitis was suggested on the plain roentgenograms and confirmed by barium enema study. She responded well to conservative management.

DISCUSSION

In the majority of our cases of advanced ulcerative colitis which were reviewed, the disease process could be recognized on the plain roentgenogram by gas delineation of the rigid tubular colon. It seems reasonable that since one of the signs of chronic ulcerative colitis is failure of the colon to collapse completely during the evacuation phase of the barium study, it should follow that this fibrotic colon would be unable to propel or expel gas as efficiently as the normal colon. The same situation should prevail where the dysfunction of the colon is associated with cathartic habituation since these two conditions may be indistinguishable by barium enema. As in most situations where hollow visceral pathology is suggested by the gas silhouette of the organs (*e.g.*, carcinoma of the stomach or colon), the impression must usually be veri-

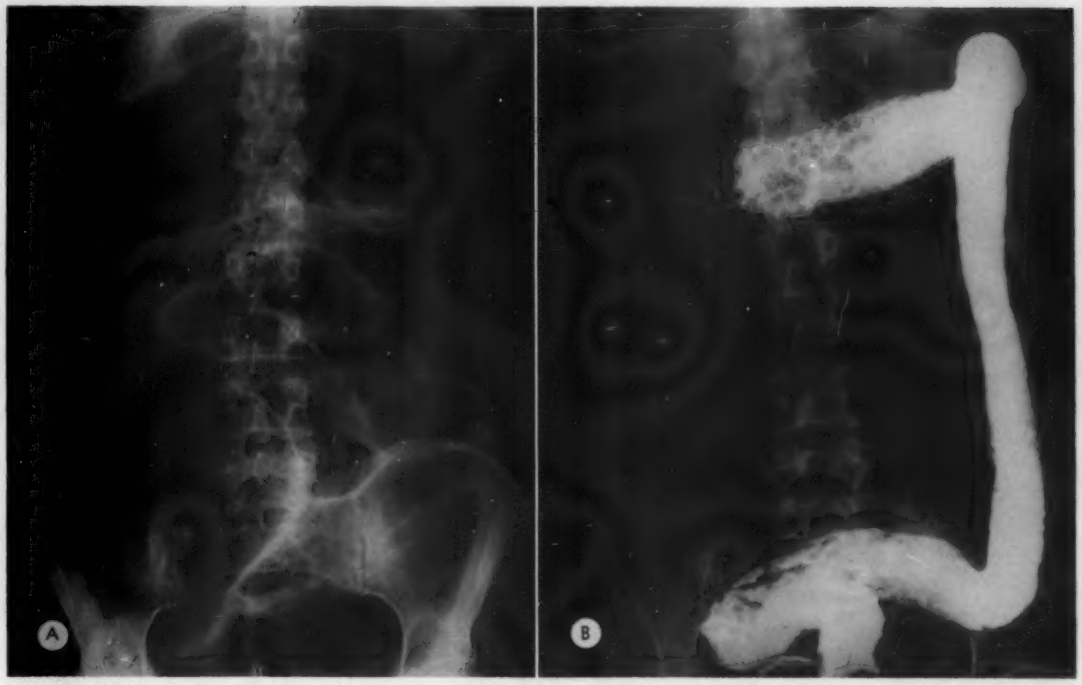

Fig. 8. Case VIII. Obstruction or fulminating colitis. (*A*) August 13, 1957. Plain roentgenogram. The distended transverse colonic segment shows thickening of the wall and nodular mucosal hyperplasia. The small bowel is also distended. (*B*) September 26, 1957. Barium enema study. The distal segment filled via rectum as far as the colostomy. The disease is apparently active with mucosal ulcers and very prominent inflammatory pseudopolyps. The inflammatory obstruction seems to have relented.

fied by opaque contrast examination.

Conversely, in acute ulcerative colitis without fibrosis of the wall where there is no loss of the haustral pattern and no interference with contractile function of the colon, one would expect that the irritability and spasm would not permit retention of gas. Our experience supports this hypothesis.

In the diagnosis of fulminating ulcerative colitis, aside from the observation of deficient haustral pattern in the colon, the primary factor seems to be an ileus or gaseous distention involving the small bowel or all of the gastrointestinal tract. Dilatation of the transverse colon, while usually present, may not be striking. The other features as described by McConnell *et al.*,[4] such as thickening of the colonic wall and nodular mucosal hyperplasia, may not be apparent, but, if seen, lend support to the diagnostic impression.

We would not anticipate frequent recognition of regional enteritis from the plain roentgenograms since the small bowel usually does not contain significant amounts of gas and the involved areas rarely produce obstruction. Deformity of the cecum and ascending colon, as occurred in our cases, is not unusual where regional enteritis involves the terminal ileum[1,3] and is due primarily to the inflammatory process in the mesentery and pressure of the thickened distal ileum. We are unaware of any other cases of regional enteritis diagnosed from the plain roentgenogram.

SUMMARY AND CONCLUSIONS

1. Late stage ulcerative colitis can often be recognized from the plain roentgenogram by the delineation of an abnormal colonic silhouette by intraluminal gas. This is not confined to fulminating cases or those with obstruction.

2. The bowel of chronic laxation may

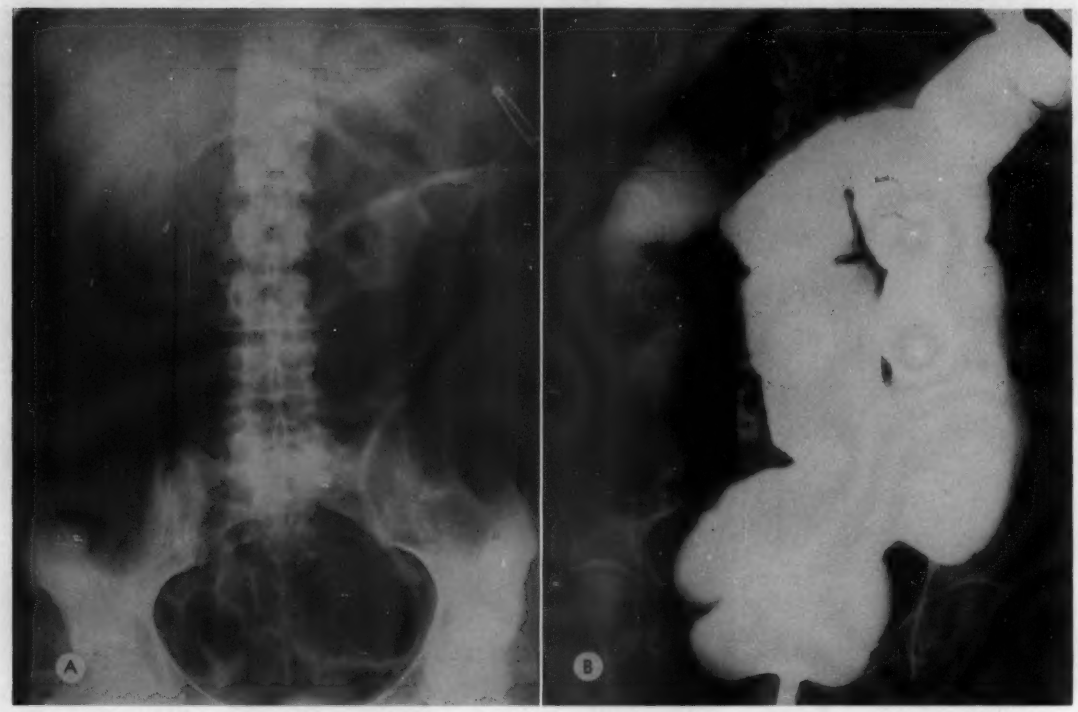

FIG. 9. Case IX. Probable cathartic bowel. (*A*) Plain roentgenogram. Characteristic absence of haustration throughout the colon. (*B*) Barium enema study. Generally capacious colon.

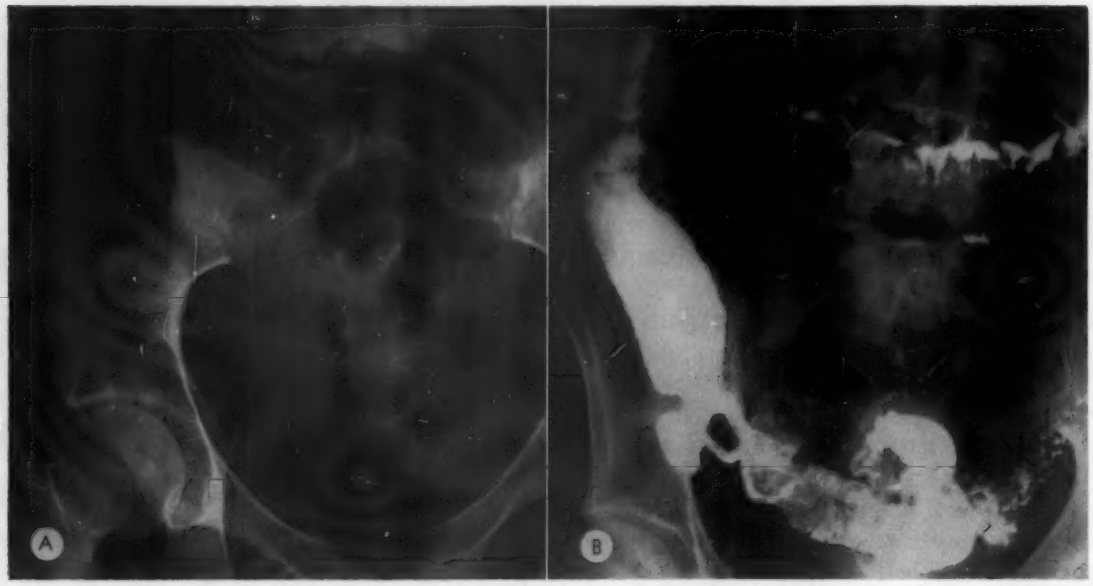

FIG. 10. Case X. Terminal ileitis. (*A*) Plain roentgenogram. The intestinal gas sharply outlines the narrow irregular lumen of the terminal ileum and the accompanying cecal deformity. (*B*) Barium enema study. Duplication of silhouette by positive contrast. The artifacts are due to mutilation of the film during automatic processing.

exhibit a similar appearance to that of late ulcerative colitis but it would be difficult to differentiate these two conditions on plain roentgenograms as satisfactorily as by barium enema study.

3. Acute or early ulcerative colitis usually cannot be identified on plain roentgenograms.

4. The combination of an abnormal gas-outlined colon, not necessarily grossly dilated, plus gaseous distention of the small bowel should suggest the possibility of fulminating ulcerative colitis.

5. A bizarre case of chronic ulcerative colitis with colonic dilatation and fecal aggregates simulating giant pseudopolyps is presented.

6. A single case of regional ileitis diagnosed on plain roentgenograms is also presented.

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DIAGNOSTIC ACCURACY OF THE BARIUM ENEMA STUDY IN CARCINOMA OF THE COLON AND RECTUM*

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EARLY diagnosis is an immediate, logical objective in attempts to decrease the morbidity and mortality from carcinoma of the colon. Since an early lesion implies small size, considerable attention has been given to the refinement of roentgenologic procedures for the detection of tumors 1 cm. or less in diameter.[4,6,7,9,11,13,15,17,18] Welin[18] found an incidence of 11 per cent of polyps in 7,561 barium enema examinations, although only 254 (3.3 per cent) were confirmed by surgery. Such a performance does suggest that the barium enema study, when properly performed and interpreted, can lead to the detection of quite small tumors. However, this performance is exceptional, as Stevenson and Wilson[16] found an over-all incidence of 1.9 per cent of polyps in 1,500 examinations, with an incidence of 5 per cent in those patients with bleeding or a history of polyps and 0.4 per cent in asymptomatic patients. Other experienced observers[4,6,9,11,13,17] report the detection of polyps of the order of 1 cm. in diameter, although we have no reliable information as to the number missed.

The majority of cancers of the colon, and particularly those that produce symptoms, are relatively gross lesions and are much larger than 1 cm. Thus, they should be easily detected by barium enema study and it might be assumed that the incidence of detection by this method would approach 100 per cent. In daily practice, however, it is doubtful that this accuracy obtains. Schutt and Walker[14] give two reasons for delay in the diagnosis of colonic cancer, one of which is "the unquestioned acceptance of negative x-ray findings." They also mention "diagnostic lassitude engendered by a series of negative colon x-rays. . . ." Ramsay[12] found that the radiologist using the barium enema study failed to detect the lesion in 10 of 75 cases of carcinoma of the colon, causing a considerable delay in the treatment of 3 cases. In 50 consecutive cases of carcinoma of the cecum and ascending colon, Brown and Colvert[1] found that the radiologist failed to detect the tumor in 4 cases, although 2 or more barium enema studies were done on 10 of these cases. Only 36 of these 50 lesions were detected at the first examination.

Case[3] stated that the well-trained radiologist should identify malignant tumors of the colon, exclusive of the rectum, in 90 per cent of the cases, and Carman and Fineman[2] found their accuracy in 359 cases to be about 90 per cent. None of the reports of the accuracy of the barium enema study, save those of Haggie,[10] have included a concerted attempt to follow up all of the supposedly negative barium enema examinations performed during the period of study. Haggie[10] found 15 cancers in 227 examinations, with 153 examinations being reported as entirely negative. However, a follow-up for a two year period showed an additional case of carcinoma of the colon which was presumed to have been missed. The number of cases involved in this study was too small to permit definite conclusions. Furthermore, the labor necessary for follow-up of the thousands of barium enema examinations done every year in a busy department makes one skeptical of the justification of such an effort.

We have not been able to find any investigations which deal with the incidence of false positive diagnoses of carcinoma of the colon.

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PURPOSE OF STUDY

The present study was undertaken to determine the accuracy of the barium enema examination in the diagnosis of carcinoma of the colon in our own institution, the reasons for failure, and to seek means for improving our accuracy.

METHOD OF BARIUM ENEMA EXAMINATION

Cleansing of the colon was routinely attempted by the administration of two ounces of castor oil about fourteen hours preceding the examination, or at about 7:00 P.M. in the evening. Early the next morning, and at least two hours preceding the barium enema, tap water enemas were given until the return water was clear. The patient did not take anything to eat or drink after midnight preceding the examination. Adequate cleansing of the colon remains something of a problem in our institution, although, as will be seen, poor preparation was only a minor contributing cause of false negative errors in gross carcinoma of the colon.

During the first five years of the study, barium sulfate from a number of different manufacturers was mixed by volume with tap water to form a suspension "somewhat thinner than stomach barium." From the roentgenograms, it appears to have been more dense than the suspension we are now using. During the last twenty months of the study, the barium was carefully weighed to produce a mixture of 1:5 of barium and water. In addition, tannic acid 1 per cent and suspending and defoaming agents were also added. For barium-air contrast studies, 800 gm. of StaBarium* was mixed in 2,000 cc. of tap water to which 2 cc. of antifoam was added.

Barium was instilled under fluoroscopic control and spot roentgenograms were exposed according to the desires of the examiner, although a minimum or basic spot roentgenographic procedure was routine. After filling the colon, and, if possible, the terminal ileum, one or more large roentgenograms of the colon were made, the patient was allowed to evacuate, and again one or more large films were exposed. During the first five years of the study, fluoroscopy and spot roentgenography were carried out after evacuation. However, during the last twenty months of the study, this was done only in selected cases.

In addition to the basic, or routine, examination just described, 27 patients underwent the barium-air contrast examination and 2 of these examinations were described as unsatisfactory. This examination was most frequently performed by instilling barium for a variable distance, usually to the region of the splenic flexure, followed by administration of air without evacuation, although at times a small evacuation was allowed so as to properly adjust the amount of barium in the left side of the colon and rectum. During the instillation of air, the patient was placed in varying positions on the abdomen and side and in the Trendelenburg position in order to distribute the barium evenly over the inside of the colon.

A review of the roentgenograms shows that all positions and projections were used at one time or another in the series. These included the lateral view of the sigmoid and the obstetrical view. Stereoscopic roentgenograms were made in a number of instances.

The examinations were carried out by approximately 20 different individuals, 7 of whom were members of the senior staff of the Department and the remainder were residents with six months to three years of training in this field. In most instances, the roentgenograms of the patient examined by the residents were also reviewed by the senior staff of the Department. It is evident that there was some variation in the technique and thoroughness of the examinations over the period of study. We believe, however, that this variation is rather typical of the average large department of radiology in which a considerable number of residents are trained.

* Distributed by Keleket X-Ray Corporation.

CRITERIA AND METHOD OF EVALUATION

Unfortunately, as is the situation in most other studies of this nature, we were limited to the false negative reports. We personally know of false positive reports given during the period of study, some of which resulted in useless surgery. Nevertheless, our indexing system and records do not give us a ready means of finding such reports, so that a precise evaluation is impossible.

False negative cases came to our attention in three ways: (1) demonstration of a lesion by barium enema after a significant delay (one month), following a previously presumed negative barium enema examination; (2) the finding of a cancer by proctoscopy, surgery, or autopsy within two years of our negative study under circumstances indicating that the lesion was overlooked; and (3) reports from referring physicians that a lesion was found and verified elsewhere within a period of two years after our negative study. If a lesion was missed on a first barium enema examination and detected on a second examination which was requested immediately by either the radiologist or the referring physician, this was not classified as an error. Delay in treatment was considered to exist when the correct diagnosis was not clearly stated and, as a result, treatment was not instituted for a period of one month or longer. This eliminates all administrative delays and those necessitated by the poor medical condition of the patient. Using these criteria, it is obvious that the interpretation is the best that the figures will permit.

MATERIAL AND METHOD OF STUDY

Our patients came from a 950-bed general hospital with a moderately active outpatient department. About two-thirds of the patients were indigent or part-pay and about the same proportion of barium enema studies were performed by residents under the supervision of the senior staff. The senior staff personally performed the remainder of the examinations.

Table 1

CARCINOMA OF THE COLON AND RECTUM SEEN IN THE UNIVERSITY OF TEXAS MEDICAL BRANCH HOSPITALS FROM JANUARY 1, 1950 THROUGH AUGUST 31, 1956

Consecutive Cases	249
Polypoid Lesions with No Invasion of Stalk	21
Invasive Carcinoma	228

Table 1 shows that 228 cases of microscopically verified, invasive carcinoma of the colon were available for study. An additional 21 cases with polypoid lesions without invasion of the stalk were eliminated. Multiple lesions occurring during the period of study were not counted as separate cases. The hospital records of the cases were examined as to presenting symptoms, duration of illness, roentgenologic report, treatment, surgeon's and pathologist's descriptions of the tumor, and subsequent follow-up. Although roentgenograms of most of the cases were available and were subjected to careful study, this retrospective information was not used in evaluating the accuracy or yield of the barium enema study. It was, however, used in assessing the reasons for failure.

EVALUATION OF DATA

Figure 1 shows the distribution of the tumors, which closely resembles that found

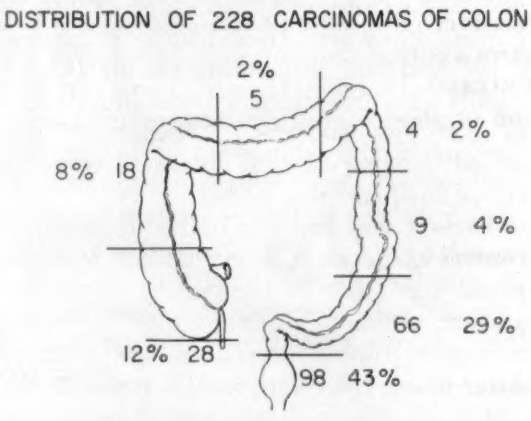

Fig. 1. Distribution of tumors.

TABLE II

INCIDENCE

Total No. of Cases with Barium Enema Studies	179
Approximate No. of Barium Enema Studies Performed during Period of Study	12,250
Examinations Performed per Carcinoma	68/1
Incidence of Carcinoma	1.5%

by most investigators. Table II shows that 179 patients in the group had one or more preoperative barium enema examinations and during the period of study approximately 12,250 barium enema examinations were performed, giving an incidence of carcinoma of 1.5 per cent. We could not find comparative figures in this regard, other than those of Christie *et al.*[4] who found 38 carcinomas in 4,225 barium enema examinations. It would be interesting to determine the effects of the low incidence of positive findings on the diagnostic accuracy of the barium enema examination. Certainly, a high percentage of negative examinations tends to dull the interest of the radiologist.

Table III shows that with the barium enema examination, a correct diagnosis was made in 103 instances, whereas the diagnosis was made by proctoscopy and finger in 109 cases and by surgery and autopsy in 16 cases.

Table IV shows that when the rectum is excluded, the barium enema study becomes relatively much more important. In these cases a correct diagnosis was made in 103 of 130 cases. In the total of 109 cases in which one or more barium enema studies were done, the lesion was overlooked in 11 instances, although it was later diagnosed correctly in 5 of these cases. This gives an over-all false negative error of approximately 10 per cent.

TABLE III

DIAGNOSTIC MODALITY

Diagnosis by:	Barium Enema Study	103
	Proctoscope and Finger	109
	Surgery	15
	Autopsy	1
Total		228

TABLE IV

DIAGNOSTIC MODALITY EXCLUSIVE OF RECTUM

Exclusive of Rectum	130
Having One or More Barium Enema Studies	109
Diagnosed by Barium Enema Study	103
Missed by Barium Enema Study (5 later diagnosed by this means)	11
Diagnosed by Surgery	15
Diagnosed by Proctoscopy	11
Diagnosed by Autopsy	1

DIAGNOSTIC ACCURACY IN SPECIFIC SEGMENTS

Cecum and Appendix

Table V shows that of 22 cases the lesion was missed in 6, giving a false negative error of 27 per cent. In only 3, however, did the diagnostic failure cause a delay in treatment.

ILLUSTRATIVE CASES OF SIGNIFICANT DELAY IN TREATMENT

CASE 1 (Fig. 2, *A*, *B* and *C*). This fifty-one

TABLE V

CARCINOMA OF APPENDIX AND CECUM

Total Cases	28
Barium Enema Study Performed	22
Diagnosed Correctly	16
Missed (2 subsequently diagnosed by barium enema study)	6
Diagnosed by Surgery (4 missed by barium enema study)	10
False Negative Error	27%

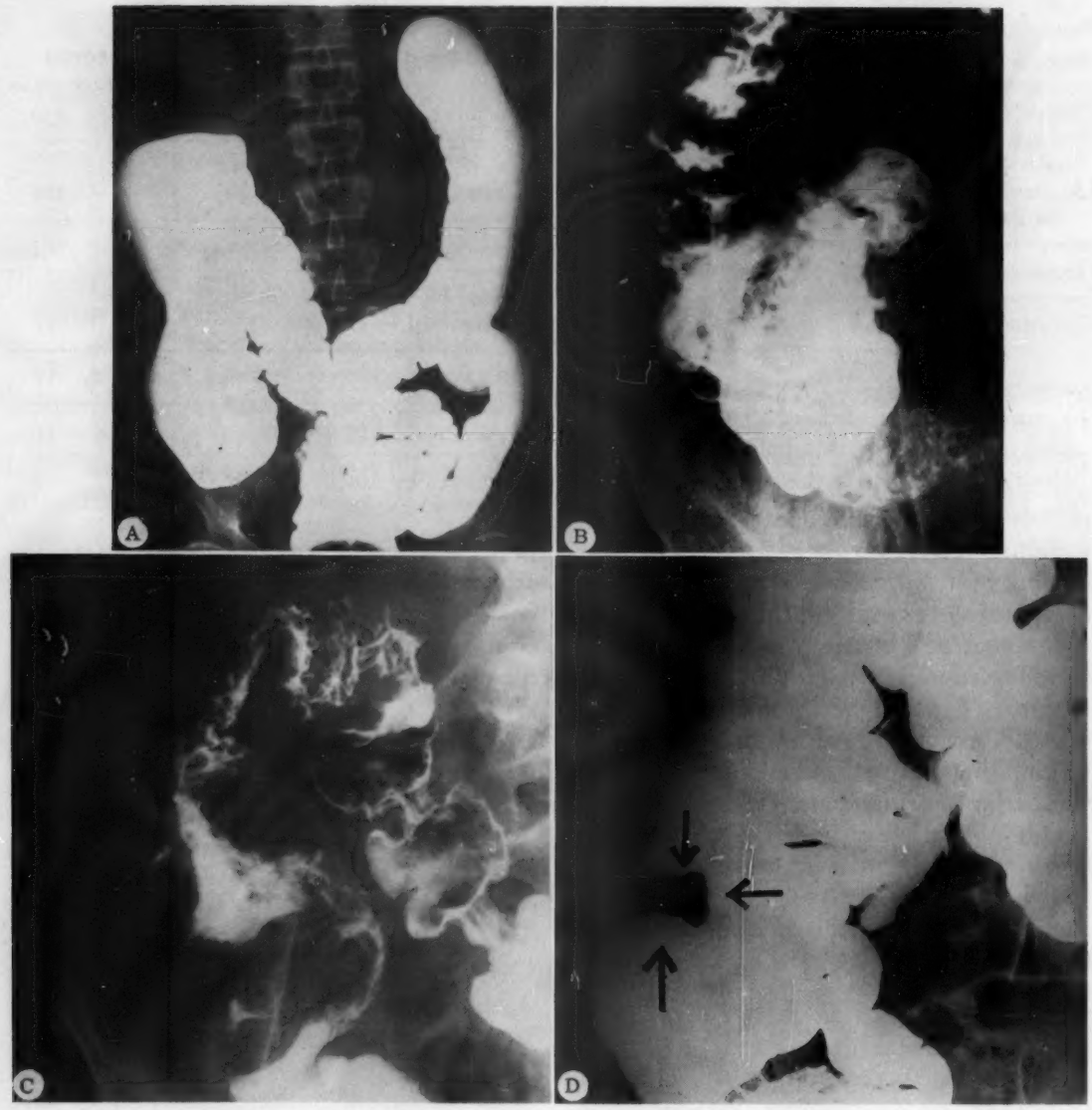

FIG. 2. Case 1. (*A*) Cecum appeared to fill adequately and was normal in contour. (*B*) Evacuation was incomplete and mucosal pattern was obscured. (*C*) One year after *A* and *B*, the evacuation roentgenogram showed a large tumor with obliteration of the normal mucosal pattern. A palpable mass was felt. (*D*) (From another patient.) A 3 cm. polypoid carcinoma on the lateral wall of the cecum (arrows). This was the only instance in this series in which a carcinoma occurred primarily in this location.

year old white male had complaints of severe anemia and constipation. The initial examination on November 17, 1953 showed an apparently filled cecum, normal in contour (*A*), but evacuation was not complete (*B*), so that an adequate mucosal study was not obtained. The patient returned approximately one year later with a palpable right lower quadrant mass and a large, easily demonstrable defect in the cecum with extrinsic pressure on the terminal ileum (*C*).

Comment. We cannot be certain that the cecum was completely filled (*A*). The evacuation roentgenogram (*B*) did not present a good mucosal study. This could have been remedied by compression spot roentgenograms after evacuation, although good mucosal detail can often be obtained by spot roentgenography during filling of the cecum. The diagnostic error here should be attributed to a low index of suspicion

and failure to obtain an adequate technical study.

Case II (Fig. 3, *A*, *B*, *C* and *D*). This sixty-eight year old white male complained of mid-epigastric pain and had a proved gastric ulcer. An upper gastrointestinal series (*A*) and barium enema studies (*B* and *C*) were done in November, 1954. A five hour roentgenogram following the gastrointestinal series (*A*) showed no gross cecal lesion, but this examination is notoriously unreliable. The barium enema (*B*) again obscured the lesion, but the evacuation roentgenogram (*C*) clearly showed a polypoid, intraluminal tumor, which was apparently not recognized. The patient returned in May, 1955 and a barium enema study (*D*) showed a gross tumor that had been neglected for six months.

Comment. The diagnostic error here seems to have been due to failure of the observer to notice a relatively small, but obvious, lesion in the evacuation film (*C*). The examiner was generally a very careful observer and this error appears to have been purely a breakdown in the normal habits of observation. At the same time, the clinician was probably lulled by the associated gastric ulcer.

Case III (roentgenograms have been discarded). This fifty-eight year old white female passed a cupful of bright red blood by rectum and came immediately to the hospital. Bleeding ceased after two days, following which conventional and barium-air contrast enema studies together with a gastrointestinal series, were carried out on successive days by the same examiner. No lesion was found, although a small shadow was seen in the cecal area. A second examination was performed and the original shadow could not be seen and was, consequently, thought to be due to fecal material. No more bleeding occurred until five months later, when the patient returned as an emergency case because of massive bleeding. A mass was easily felt in the right lower quadrant and surgery was performed immediately, revealing a 3 cm. long, annular, constricting carcinoma of the cecum.

Comment. The shadow in the cecum, ascribed to fecal material on the initial examination, may well have been the tumor. The second examination, however, failed to reveal the shadow, even though particular attention was given to this area.

Illustrative Cases Without Significant Delay in Treatment

Case IV (Fig. 4, *A* and *B*). This sixty-six year old white male had experienced lower abdominal pain for four months prior to admission. A barium enema study was requested because a small polyp was found in the rectum at protoscopy. On admission, the hemoglobin was 14.6 gm. A 1.5 cm. polyp with a long pedicle was detected in the sigmoid, which was clearly visible on the spot roentgenogram (inset *A*). Barium did not pass into the terminal ileum, but the fluoroscopist thought that the cecum had filled. A barium-air contrast enema study on the following day showed the polyp (*B*). The cecum had somewhat the same appearance as on the previous day and was certainly not adequately distended, suggesting an obstruction to retrograde flow. Gas was also detected in the terminal ileum, suggesting an early obstruction. Operation a few days later confirmed the diagnosis of a polyp in the sigmoid, but a large mass was found in the cecum and there were numerous implants in the peritoneal cavity. Tissue removed for biopsy showed an adenocarcinoma and the tumor was inoperable.

Comment. The examiner devoted much time to the polyp and failed to appraise the appearance of the cecum carefully. Both incomplete filling and incomplete emptying of the cecum may obscure a lesion.

Case V (Fig. 5, *A*, *B*, *C* and *D*). This seventy-one year old white female, two months prior to admission, began to expel small amounts of dark and bright red blood, associated with mild diarrhea. Two weeks before admission, a barium enema study done elsewhere (*A*) showed an irregular polypoid shadow that could represent a tumor, although the cecum had a normal contour otherwise. The hemoglobin was 3.2 gm. and the stools were repeatedly positive for occult blood, with an occasional stool showing gross dark red blood and mucus. Illustrations *B*, *C* and *D* were selected from three consecutive barium enema examinations performed during a ten day period. *B* shows a spasmodic peristaltic wave with almost complete emptying of an irritable cecum, and the wall appears flexible and mobile. *C* shows the cecum to be

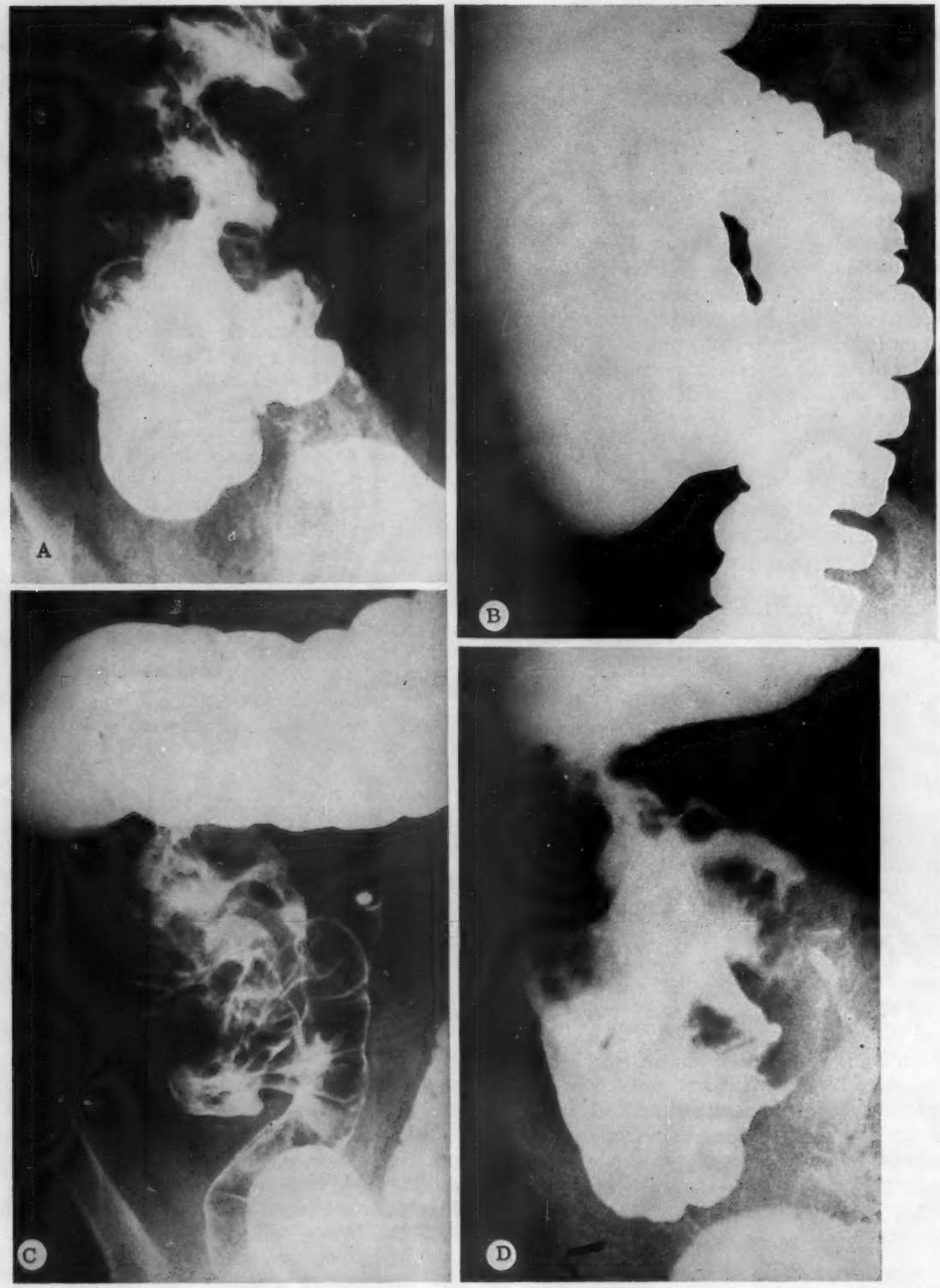

FIG. 3. Case II. (*A*) Five-hour roentgenogram following an upper gastrointestinal series. The cecum appeared grossly normal, but a roentgenogram such as this is inadequate and should be disregarded. (*B*) Cecum was presumably filled, showing no deviation in contour. (*C*) Cecum evacuated satisfactorily, but rounded tumor mass was either overlooked or disregarded. (*D*) Six months later, a large, polypoid tumor was visualized.

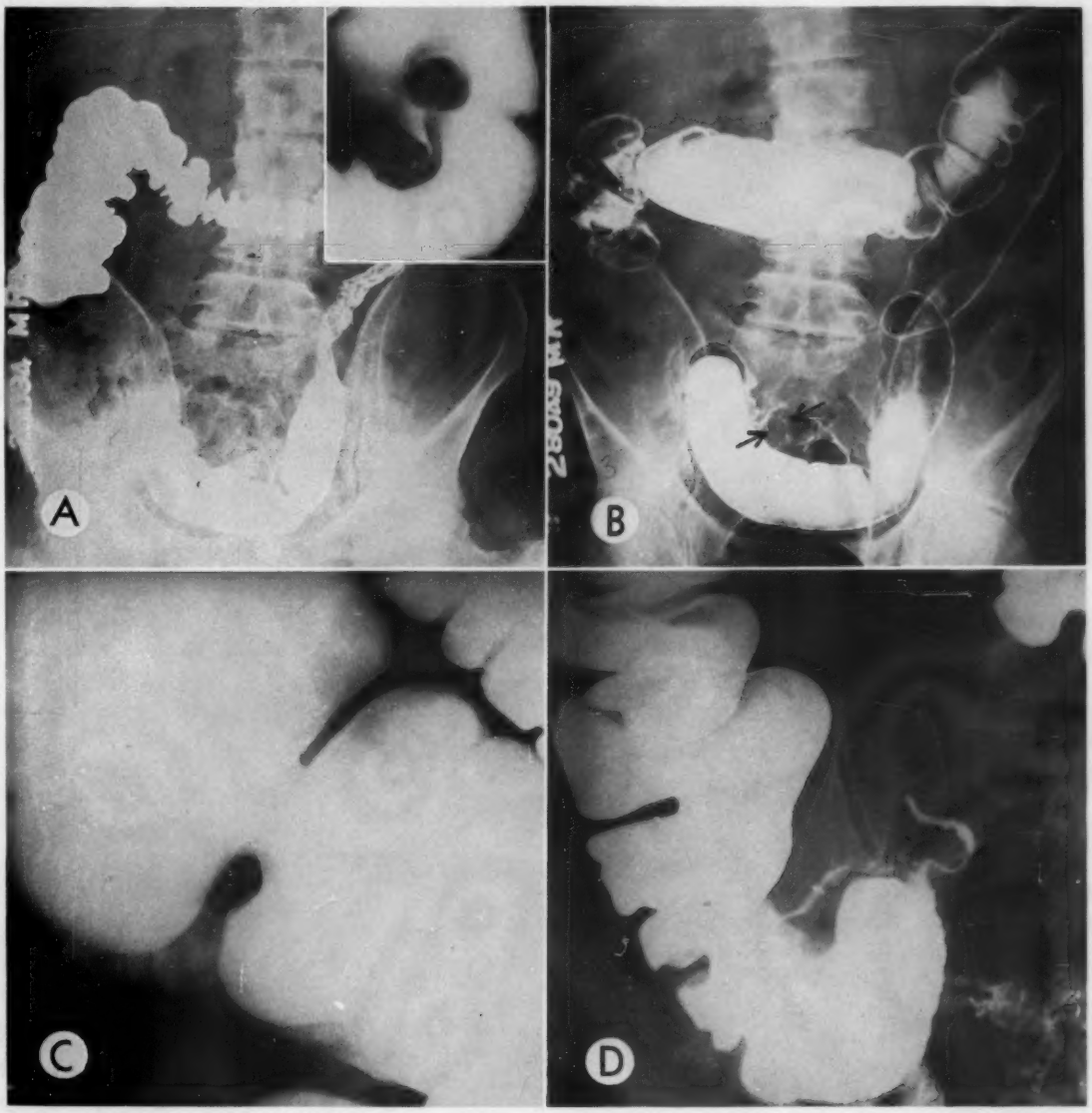

Fig. 4. Case IV. (*A*) Examination was done because of rectal bleeding. Fluoroscopist saw a polyp in sigmoid (inset), but failed to note sharp margin of barium just above the cecum. (*B*) Barium-air contrast enema study performed the following day. Again, the cecum was not adequately distended. The polyp in the sigmoid is easily seen. At surgery a few days later, a large, inoperable carcinoma of the cecum was found. (*C* and *D*) (From another patient.) Annular carcinoma near ileocecal valve about 3 cm. in length (see text).

moderately distended and the contour is slightly irregular, but there is uncertain evidence of an intraluminal mass. *D* shows the cecum maximally distended after using a double contrast technique. Comparison with the barium enema study performed five weeks previously (*A*) suggested that considerable scarring and contracture had occurred during the interval. The radiologist felt that a tumor was unlikely and the pericecal adhesions might be the cause of the deviation in the cecal contour. At operation a few days later, a small mass—3 cm. in diameter—was found in the posterior wall of the cecum, 4 cm. above the base of the appendix. The pathologist found puckering of the serosa at the site of the tumor. Upon opening of the resected specimen, the lesion with a flat surface measuring 3×2.5×0.8 cm. protruded only slightly into the lumen. The mucosa over the lesion was not ulcerated, nor

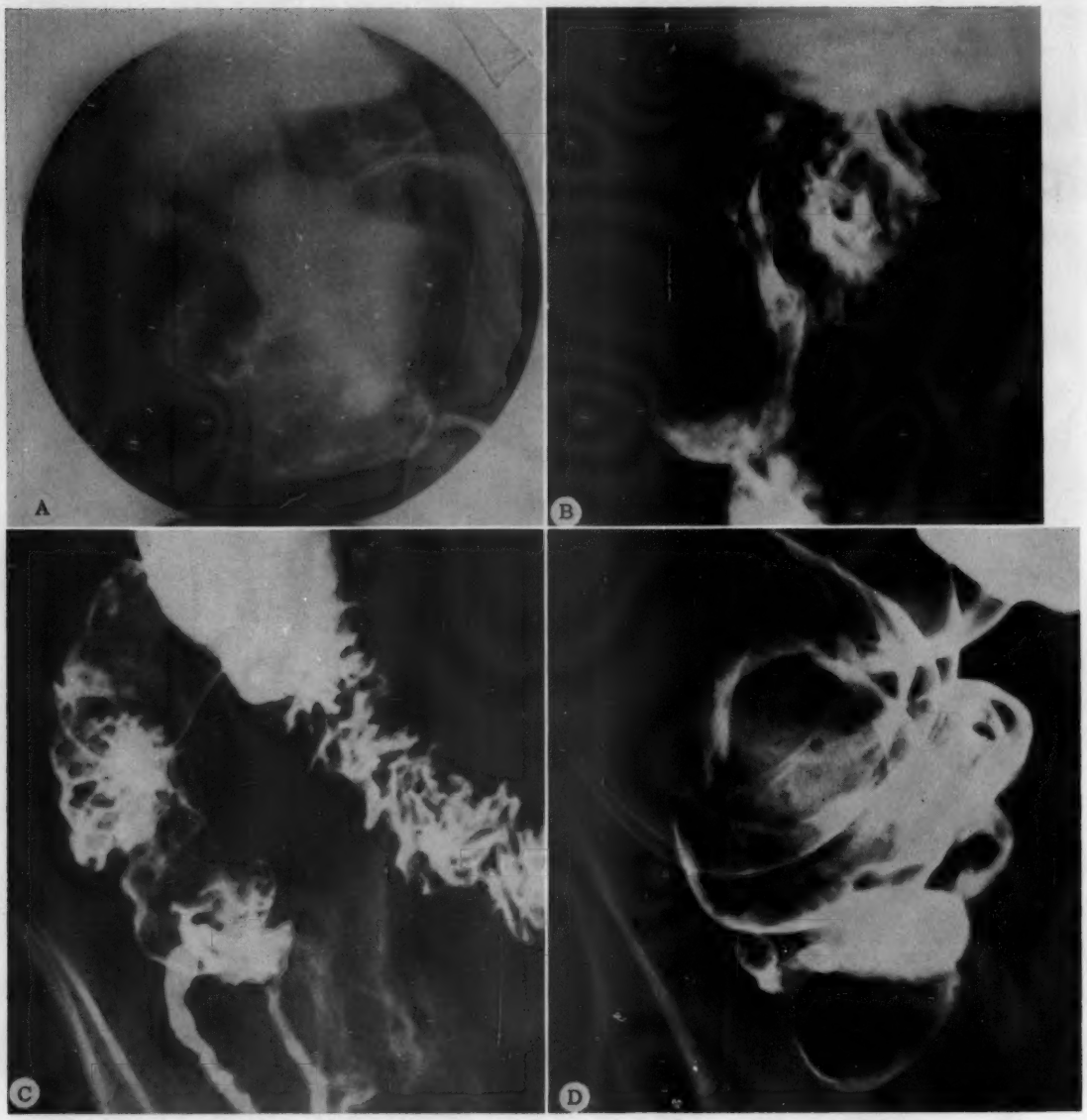

FIG. 5. Case v. (*A*) Polypoid radiolucent area on lateral and posterior wall of cecum was interpreted as a carcinoma by outside radiologist. (*B*) Three weeks later, roentgenogram taken at height of a peristaltic wave showed good evacuation and no evidence of a tumor. (*C*) Evacuation roentgenogram from a subsequent examination showed slight irregularity in contour, but no polypoid mass. (*D*) Barium-air contrast study showed that the cecum did not distend normally. No tumor mass was seen. The changes were interpreted as being due to scarring (see text for findings at operation).

had the lesion extended significantly into the bowel wall. A benign adenomatous polyp—measuring 8×5×3 mm.—was found 4 cm. distal to the larger malignant lesion.

Comment. Despite adequate roentgenologic study, the lesion remained a diagnostic problem. Treatment was not significantly delayed, due to the patient's symptoms. The cecal contracture was out of proportion to the size of the tumor and might be compared to the incisura occasionally seen in the stomach near a small carcinoma. One could speculate that the lesion was originally polypoid and that the protruding portion had sloughed at some time between

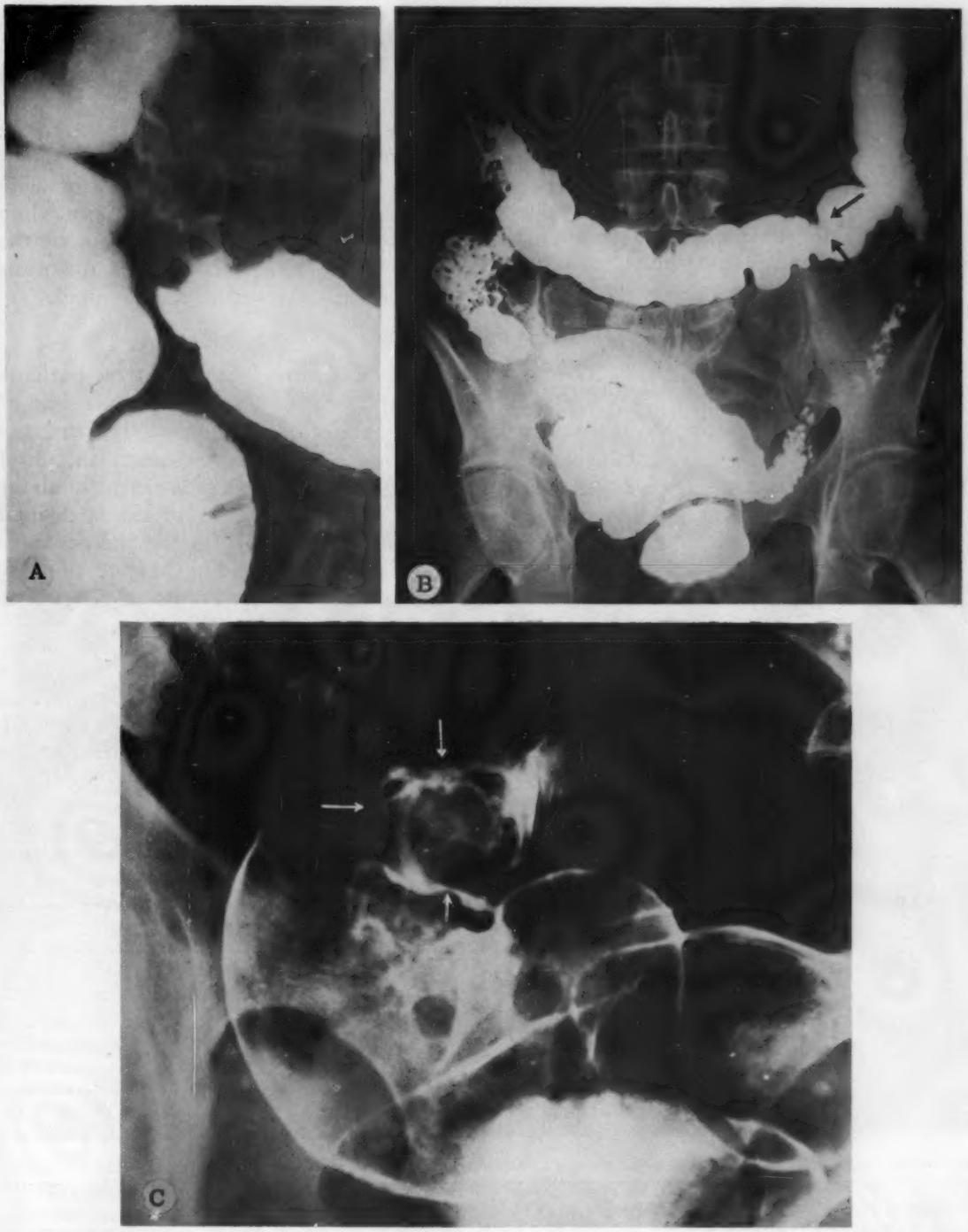

Fig. 6. Case vi. (*A*) A large tumor was demonstrated near the hepatic flexure. The mucosal pattern was distorted and the margins were sharp with typical overhang. (*B*) A 3 cm. polypoid tumor (arrows) was seen in retrospect in the transverse colon. In addition, there was a 7 mm. carcinoma in the cecum. Little attempt was made to study the remainder of the colon after the gross lesion was seen near the hepatic flexure. Case ix. (*C*) Air-barium contrast enema study showed a polyp on the left wall of the sigmoid. It was easily seen through the proctoscope. The radiologist did not mention the lesion in his report.

the first and second examinations, leaving only the tumor base.

CASE VI (Fig. 6, *A* and *B*). This sixty-five year old white male had a large tumor, 10 cm. in length, which was easily demonstrated in the right side of the transverse colon (*A*). Justifiably, the radiologist did not attempt a careful study of the cecum after observing the lesion near the hepatic flexture. (*B*) At operation, the surgeon inadvertently cut through a 1.5 cm., elevated, polypoid carcinoma in the left side of the colon (arrows) and, consequently, had to revise the resection. In retrospect, the lesion in the transverse colon is easily seen. The pathologist found another small, ulcerative carcinoma, 7 mm. in diameter, in the cecum.

Comment. Although this case is included twice in the list of negative cases (under both cecal and transverse colon tumors), the cecal tumor was an incidental finding and was not susceptible to roentgenologic diagnosis. No delay in treatment occurred.

Segment between the Cecum and Sigmoid Colon

Thirty-six cases of primary tumor of the segment between the cecum and sigmoid colon were encountered and a barium enema study was performed on 35 of these, all of which were correctly diagnosed, with the single exception of the left transverse colon lesion mentioned above. Although the total number is small, this performance confirms the impression that carcinomas in this segment, encompassing more than three quarters of the total length of the colon, are more susceptible to diagnosis than those in the cecum and sigmoid.

Sigmoid Colon

Table VI shows that 52 of 66 patients with carcinoma of the sigmoid colon had at least one barium enema study, and the lesion was correctly diagnosed in 48 of these, giving a false negative error of about 8 per cent. The sigmoid colon is defined here as that portion between the descending colon and, roughly, the line of peritoneal reflexion where the bowel penetrates the upper part of the pelvic floor.

ILLUSTRATIVE CASES

CASE VII (Fig. 7, *A* and *B*). This seventy-two year old white female was seen for a check-up and denied any complaints. A barium enema

TABLE VI

CARCINOMA OF THE SIGMOID COLON

Total Cases			66
One or More Barium Enema Studies			52
Correctly Diagnosed by Barium Enema Study			48
Missed			4
	Later Diagnosed by:	Proctoscopy	1
		Barium Enema Study	2
		Autopsy	1
Diagnosed by Surgery			3
Diagnosed by Proctoscopy			11
Both Proctoscopy and Barium Enema Study Positive			11
False Negative Error			8%

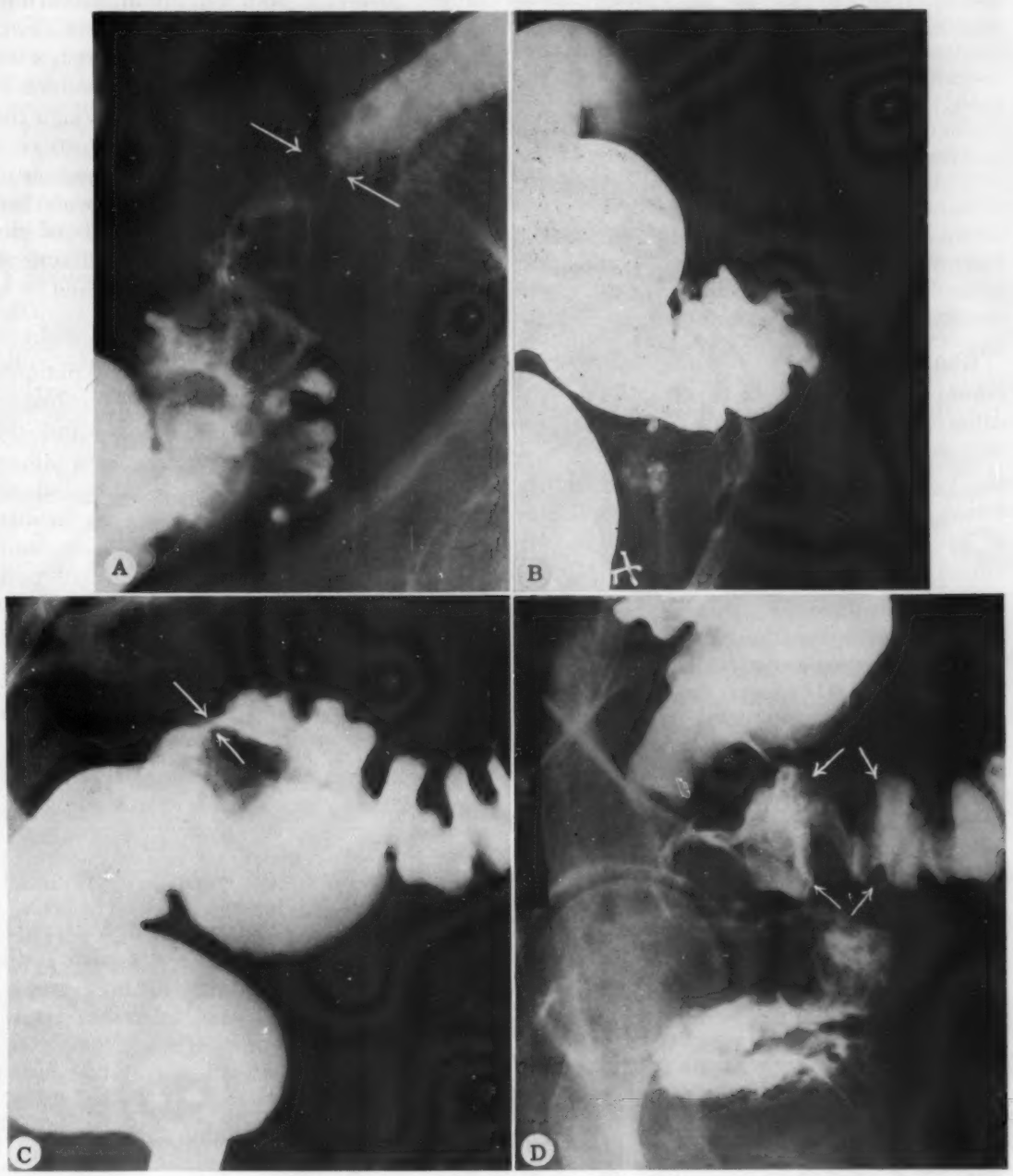

FIG. 7. Case VII. (*A*) Examination of April 9, 1956 showed a number of diverticula in the sigmoid, with some associated spasm. In retrospect, the sharp margin (arrows) might have been noted with concern. The patient was asymptomatic. (*B*) Examination of September 5, 1957 showed complete obstruction to retrograde flow and margins of the barium column were sharp with slight overhang. Case VIII. (*C*) Far oblique view made in an effort to demonstrate the sigmoid. Narrowed area (arrows) was thought by fluoroscopist to be inconstant. (*D*) Another examination a few days later showed a narrowed area, but this was thought to be inconstant and the mucosal pattern was intact. A 3 cm. annular carcinoma, 22 cm. above the anal orifice, was found at autopsy six weeks later.

study on April 9, 1956 (*A*) showed a localized cluster of diverticula in the sigmoid with some associated spasm but no local tenderness. A diagnosis of diverticulosis was made, but the possibility of a superimposed carcinoma was not mentioned. The patient returned fifteen months later with complaints of chills and fever and, in retrospect, admitted abdominal cramps. A barium enema on September 5, 1957 (*B*) showed a complete obstruction to retrograde flow with a sharp cut-off with overhanging edges. At surgery, a 5 cm. annular carcinoma was found.

Comment. The recognition of a carcinoma associated with diverticulosis, or a differentiation of these two conditions, is a recurrent roentgenologic problem. Here, a sharp margin at the upper border of the lesion (arrows) was a significant sign and should have raised the question of a carcinoma. Treatment was delayed fourteen months.

CASE VIII (Fig. 7, *C* and *D*). This forty-eight year old white male complained of right upper quadrant pain. The liver was markedly enlarged and tissue removed at exploratory laparotomy revealed metastatic carcinoma, primary site undetermined. Three successive preoperative barium enema examinations showed that the sigmoid was moderately redundant and a narrowed area was seen on several roentgenograms (*C*—arrows). This narrowing seemed inconsistent and the mucosal pattern was not grossly abnormal. Consequently, although a lesion was strongly suspected here, a positive diagnosis was not made. An air contrast examination was included, but did not demonstrate the sigmoid area to any better advantage than the conventional enema study. A small adenomatous polyp was found in the rectum at proctoscopy. At autopsy six weeks later, an adenomatous carcinoma, 3 cm. in length, was found in the sigmoid approximately 22 cm. above the anal orifice.

Comment. The lesion is probably represented by the narrowed areas (*C* and *D*—arrows), but was thought by the radiologist to be inconstant. The successive enema studies were performed by different radiologists and the constancy of the defect was either not observed or not appreciated. The failure to arrive at a correct diagnosis did not significantly affect the outcome. The lesion was not apparent to the surgeon at laparotomy.

CASE IX (Fig. 6 *C*). This sixty-three year old Negro female suffered intermittent bleeding for about one year prior to admission. A pedunculated, nodular mass was seen through the proctoscope by the patient's physician and she was referred for treatment. Two successive barium enema examinations were performed, the second employing the barium-air contrast technique. The latter examination showed the lesion to good advantage (*C*), but the radiologist made no mention of it in his report. It is presumed that he did not recognize the lesion, although he knew that a tumor had been identified through the proctoscope. A 3.5 cm., ulcerated, nodular, polypoid carcinoma was resected.

Comment. This appears to have been a failure of the radiologist to recognize a polypoid lesion in the sigmoid. In this instance, an adequate barium-air contrast examination was strikingly superior to a conventional barium enema study. There was no delay in treatment.

CASE X (roentgenograms not reproduced). This fifty-five year old white female was given a barium enema because a polyp was seen in the lower sigmoid on proctoscopy. Two successive barium enema studies, the last a barium-air contrast enema, showed no lesion. A re-inspection of the roentgenograms showed that the examination was technically satisfactory as far as the distribution of barium and air was concerned, but the sigmoid was redundant and there was a short segment which was not completely demonstrated. At operation, a 1.2×1.2×6 cm. stubby polyp was resected. It showed malignant invasion of the stalk and was classified as invasive carcinoma.

Comment. Polyps of this size are susceptible to roentgenologic diagnosis, but this requires persistent effort and often such lesions are missed.

Rectum

Table VII shows that in 34 of 70 patients with carcinoma of the rectum, the lesion was not demonstrated by barium enema

TABLE VII

CARCINOMA OF RECTUM

Total Cases	98
One or More Barium Enema Studies	70
Lesion Noted	34
Missed	36
Seen in Retrospect	2
False Negative Error	49%

examination. In reviewing the majority of these roentgenograms, it seems that the radiologist was aware of the diagnosis, but made no determined effort to demonstrate the lesion. Frequently, it was stated in the roentgenologic report that there was no lesion above the proctoscopic level. It is evident that lesions in this segment are not considered to be in the province of the radiologist and are often neglected.

DISCUSSION AND RECOMMENDATIONS

In the present study, the cecal area provided the most problems and the largest number of errors in diagnosis. Prior to this study, we had suspected that the sigmoid colon would present the greatest challenge, because of its convolutions and its relative inaccessibility to palpation. On the other hand, it is probable that the sigmoid colon receives more attention by the radiologist than the other segments, because he is aware of these difficulties. In a larger series, however, the sigmoid colon might prove to be the segment in which the largest number of errors are encountered.

Errors in the cecal area were due to failure to fill the cecum, failure to obtain adequate mucosal detail (associated, in most instances, with imperfect emptying of the cecum), failure to appreciate the varied manifestations of carcinoma in this area, or simple human failure to recognize an obvious lesion. In one case, the lesion was 8 mm. in diameter and was not susceptible to diagnosis by roentgen means.

In the sigmoid area, errors were due to incomplete demonstration of redundant loops, inability to recognize carcinoma engrafted upon a diverticulosis, or—again—human failure to recognize an obvious lesion.

The shortcomings of the radiologist may be due to inexperience, lack of enthusiasm and diligence, or plain inadequacy. However, Garland[8] has shown admirably that even trained, skilled, careful radiologists (and, for that matter, other specialists) may disagree or fail to appreciate objective evidence of abnormality. Irreducible human error will, no doubt, be with us for some time.

Descriptions of the typical roentgenologic appearance of carcinoma of the colon should be followed by the admonition that *atypical* cases are quite common. The typical lesion of the cecum is described as fungating and polypoid, whereas the sigmoid lesion is annular and obstructive. However, Figure 4, *C* and *D* demonstrates a circular, constricting lesion at the ileocecal valve, about 2 cm. in length. In the cecum, 9 of 22 cases revealed a lesion having an annular appearance; in the sigmoid colon, only 22 of the 48 cases reviewed were definitely annular in appearance. The only reliable roentgenologic sign is aberration or distortion of the mucosal pattern of the segment or direct visualization of a tumor mass.

This study has indicated to us that there is no single, certain roentgenologic approach to diagnosis of carcinoma of the colon. As Robinson[13] has stated, the procedure which may prove successful on Monday may be inadequate on Tuesday. Certainly, any rigidity of approach or absolute reliance upon any one type of barium enema examination will be, we believe, conducive to either an inordinate expenditure of labor and material or errors in diagnosis. Techniques such as described by Moreton[11] and Welin[18] doubtless would improve accuracy, but are difficult to apply to large numbers of patients. We rely upon the single contrast, or conventional enema study, using tannic acid with high voltage expo-

sures. Of course, careful fluoroscopy and high quality roentgenograms, combined with alertness on the part of the radiologist, will minimize the percentage of undiscovered lesions, no matter what technique is used.

We also believe that greater accuracy has resulted from the use of rapid developing of the roentgenograms (X-Omat), which permits a second radiologist to review all of the roentgenograms before the patient leaves the department. Fluoroscopists, after consultation with the senior radiologist who has reviewed the roentgenograms, may repeat the examination or adopt other maneuvers, such as further evacuation, spot filming of suspicious areas, injection of air, and special views, until all areas and segments of the colon have been demonstrated. This, we believe, has focused more attention on the problems of diagnosis and has led to a better technical performance.

We have also formed a teaching collection of carcinomas of the colon derived from the present study. This collection is shown at varying intervals throughout the year, particularly to the new residents, in order to demonstrate to them, in detail, the cause of certain errors in diagnosis. This has stimulated enthusiasm for the careful performance of barium enema examinations.

We believe that there is a wide difference in the care, techniques, and diagnostic yield of the barium enema study as carried out on a large scale in this country. We would prefer to believe that our performance has been average for the circumstances under which we operate regarding staff, facilities, and number of examinations. We realize that the present report is not flattering to our diagnostic achievements in the past, but we believe it has already contributed to a significant improvement in our performance of the barium enema examination.

SUMMARY

The accuracy of the barium enema examination in the diagnosis of 228 consecutive cases of carcinoma of the colon is assessed. The carcinoma was proximal to the rectum in 130 cases and in 109 of these one or more preoperative barium enema studies were performed. In this group, a tumor was overlooked 11 times, giving a false negative error of about 10 per cent. However, in only 5 cases was there a significant delay in treatment due to the roentgenologic report. Two errors occurred in a single patient who had three simultaneous tumors.

The most unsatisfactory performance was in the cecal area, where 6 of 22 cases were overlooked or misdiagnosed, although in only 3 of these cases did a significant delay in treatment occur.

In the sigmoid, 4 of 52 cases were overlooked or misdiagnosed, although in only 2 cases was there a significant delay in treatment.

Errors were due to a variety of causes, predominantly poor technique, inexperience, and momentary individual lapses. Some of the errors, we believe, were unavoidable.

Recommendations for improved performance are given.

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PROFILE LATERAL PROJECTION IN THE DIAGNOSIS OF LESIONS OF THE RECTUM*

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IT SHOULD be no secret to any experienced radiologist that the pelvic sigmoid colon and the rectosigmoid possess a highly variable anatomy which at times defies adequate visualization during routine barium enema examination and that unfortunately this same bowel segment spawns a major portion of all neoplasms of the large bowel. In an attempt to accurately diagnose lesions in this area, various projections and techniques have been introduced in the past, one of which was the lateral rectal projection employed both before and after evacuation. In the Scandinavian and most European countries this projection is considered necessary and is routinely used.[7] Previous publications by such workers as Ettinger and Elkin,[4] Robins and Altman[10] and Levene and Kaufman[6] have called attention to the advantages of this view but its use does not appear to have been fully appreciated or exploited in this country. The roentgenograms are easily and quickly obtained and require no more effort on the part of the patient than to roll into the lateral position with the knees somewhat flexed. The enema tube is removed before making the roentgenographic exposure.

The purpose of this paper is to emphasize the value of the lateral rectal profile projection in the routine barium enema study and not in any way to suggest that this view is superior to or should replace the special contrast studies of the lower colon when they are indicated and properly performed; the air contrast study of the lower colon as described by Welin[11] gives excellent mucosal and intraluminal detail and has been described as being superior to proctoscopy in some cases. In many instances, the addition of this projection to the usual series of roentgenograms provides considerable information to the radiologist regarding the condition of the upper rectum and rectosigmoid which otherwise may not be fully appreciated. In some clinics all patients referred for enema study routinely have sigmoidoscopic or proctoscopic examinations performed in conjunction with the barium studies. This is, of course, a highly commendable procedure and one which should be more generally adopted; however, in the average general hospital patients are often referred from heterogeneous sources so that it is occasionally only the radiologist who visualizes this segment of bowel. It is in this situation where the profile view probably finds its greatest value. In at least one instance in our series, a defect due to neoplasm was noted in a patient who had had a recent rectal and sigmoidoscopic examination with negative findings.

For the past eleven years, roentgenograms in this projection have been obtained routinely in all barium enema studies done in the hospitals of this area. From our files we have collected 17 cases, in each of which the lateral roentgenogram clearly and unequivocally demonstrates a neoplasm in either the rectum or distal sigmoid colon, while the remainder of the routine roentgenograms reveal little or nothing to suggest the correct diagnosis. Two such cases are illustrated (Fig. 1, *A–D* and 2, *A–D*).

At these institutions, a routine barium enema examination includes fluoroscopy totaling approximately 150 to 200 seconds with 4×6 inch shutter size, five 6½×8½ inch spot roentgenograms, four coned posteroanterior and oblique 14×17 inch roent-

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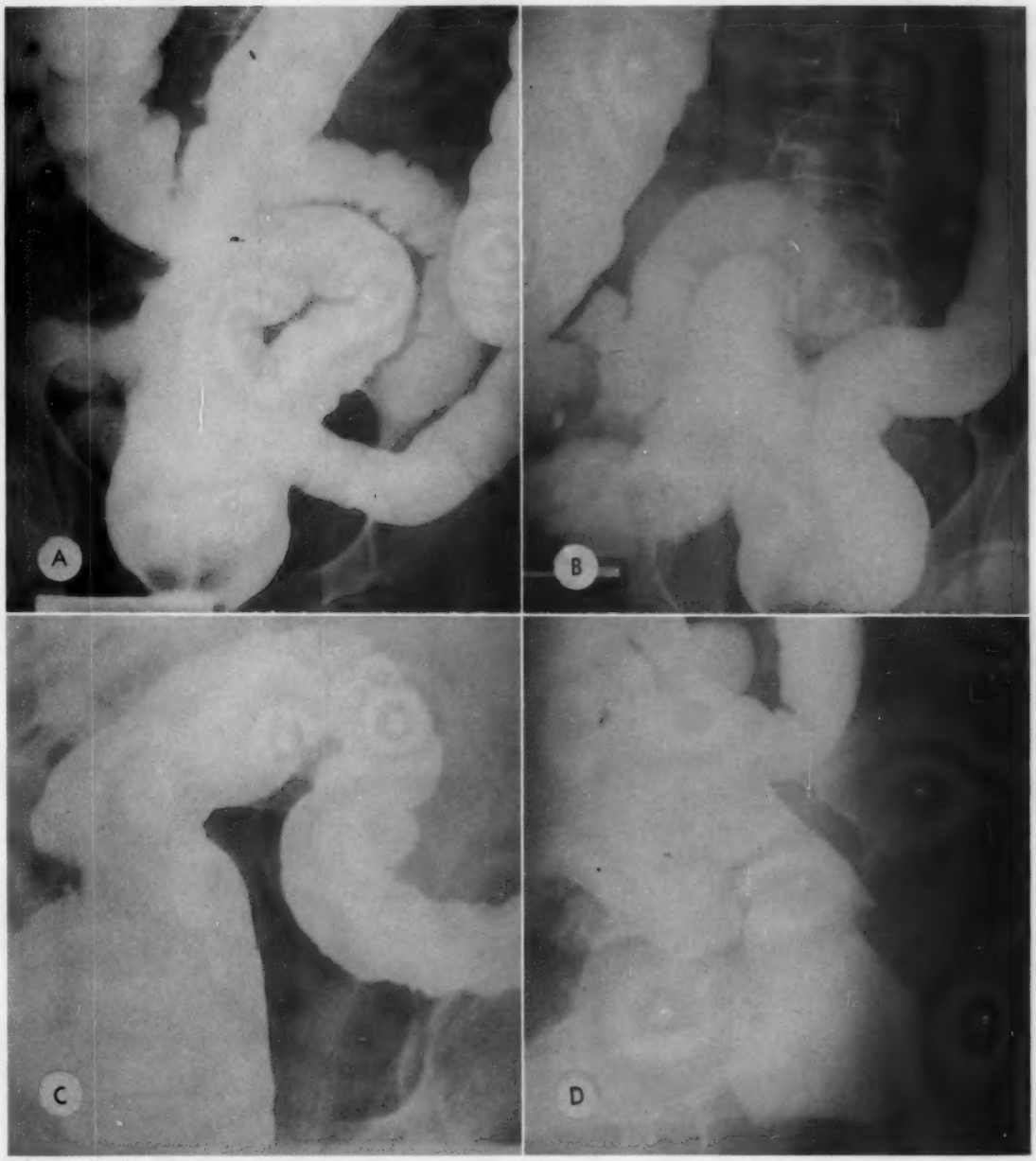

Fig. 1. (*A–D*) H.M., a fifty-six year old male, presented with a three week history of painless rectal bleeding. Barium enema study revealed an irregular filling defect on the posterior wall of the rectum seen only in the profile projection. Sigmoidoscopy and biopsy followed; a pathologic report of benign polypoid tumor was given. A resection of the polyp was then performed with a pathologic diagnosis of rectal polyp with early malignant changes.

genograms and two coned lateral roentgenograms of the rectum, one taken before and one after evacuation. It is possible, using the "retrospectoscope," to suspect minor irregularities or filling defects on some of the roentgenograms in the series other than the lateral, but without the profile view these defects might have either been completely overlooked or, if seen, their significance might not have been appreciated.

A composite collection of lateral rectal roentgenograms from the series is presented

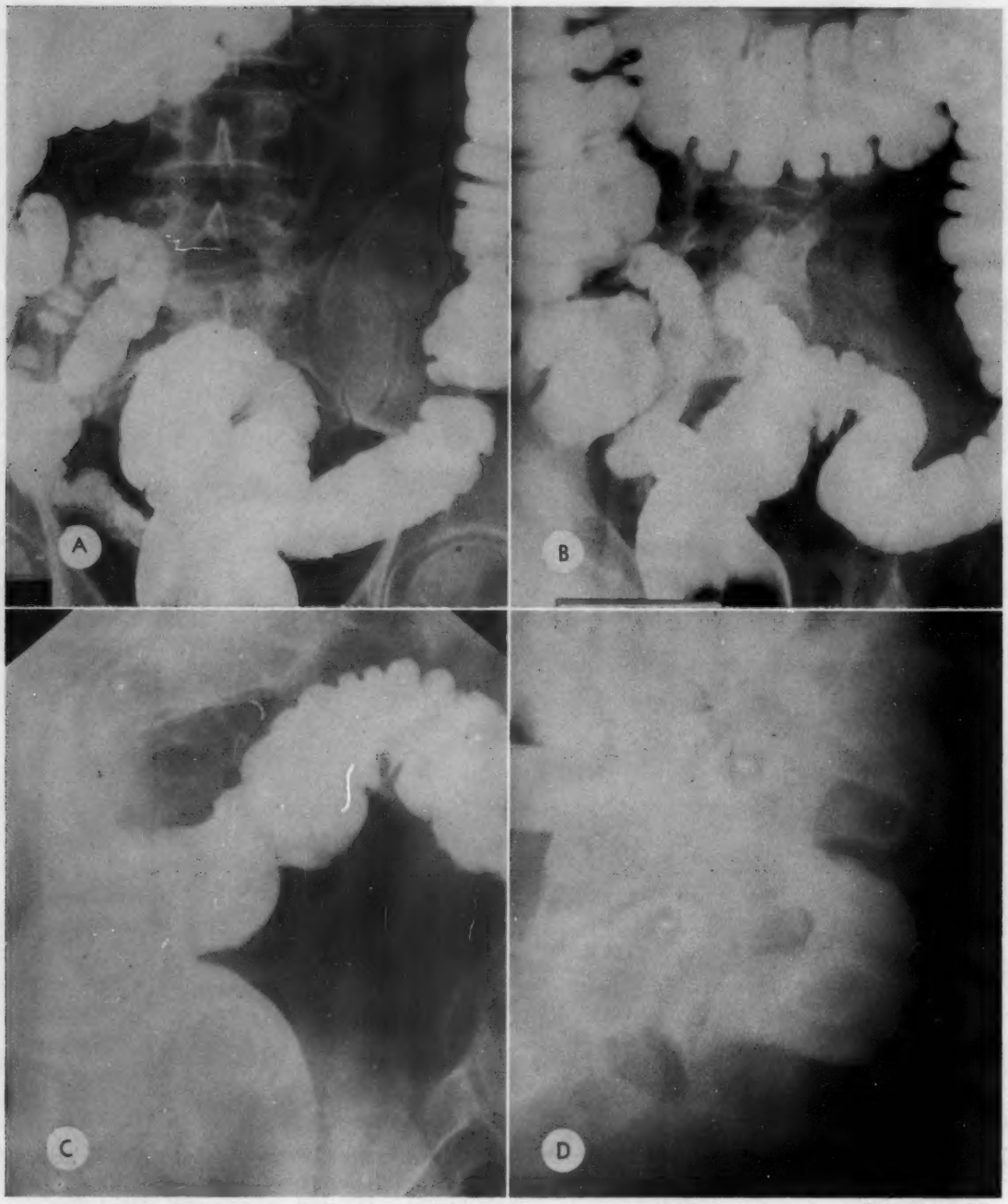

Fig. 2. (*A–D*) W.D. Previous barium enema study was reported as negative; a profile view was not used. Repeat barium study showed a long, annular irregular filling defect in the upper rectum visualized only in the lateral view which subsequently proved to be produced by an adenocarcinoma.

in Figure 3, *A–I*. This collection does not include any of that large group of cases where the lesion was clearly identified by fluoroscopy or on other roentgenograms and where the lateral view was helpful in visualizing fixation, displacement or angulation of the lower colon in association with other disease of the pelvis.

Figures 4, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B* illustrate the advantages of ob-

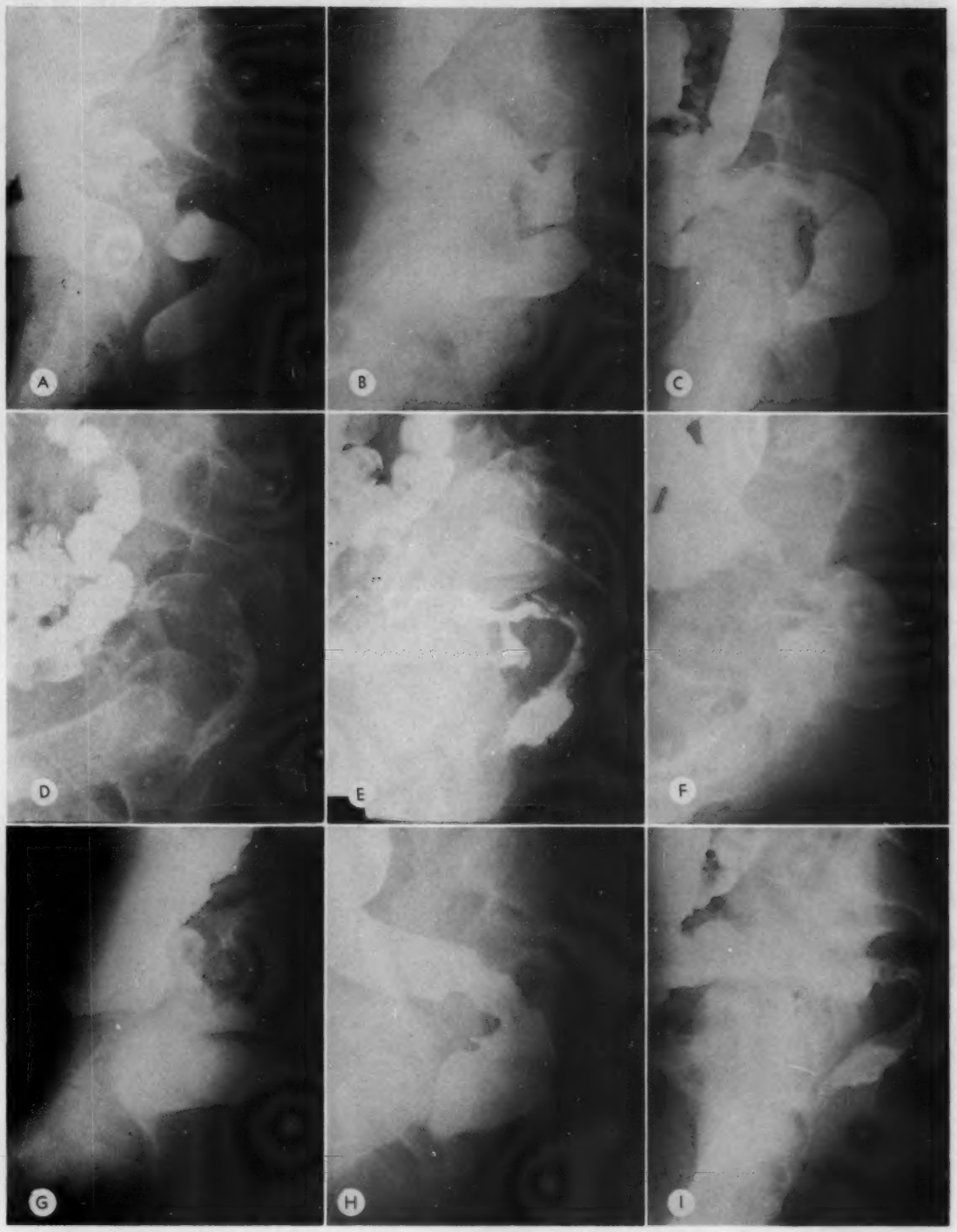

Fig. 3. (*A–I*) All of the above lateral roentgenograms of the rectum are from cases in which the lesions could not be identified on anteroposterior, oblique or "spot" roentgenograms. In all, the lesions were adenocarcinomas.

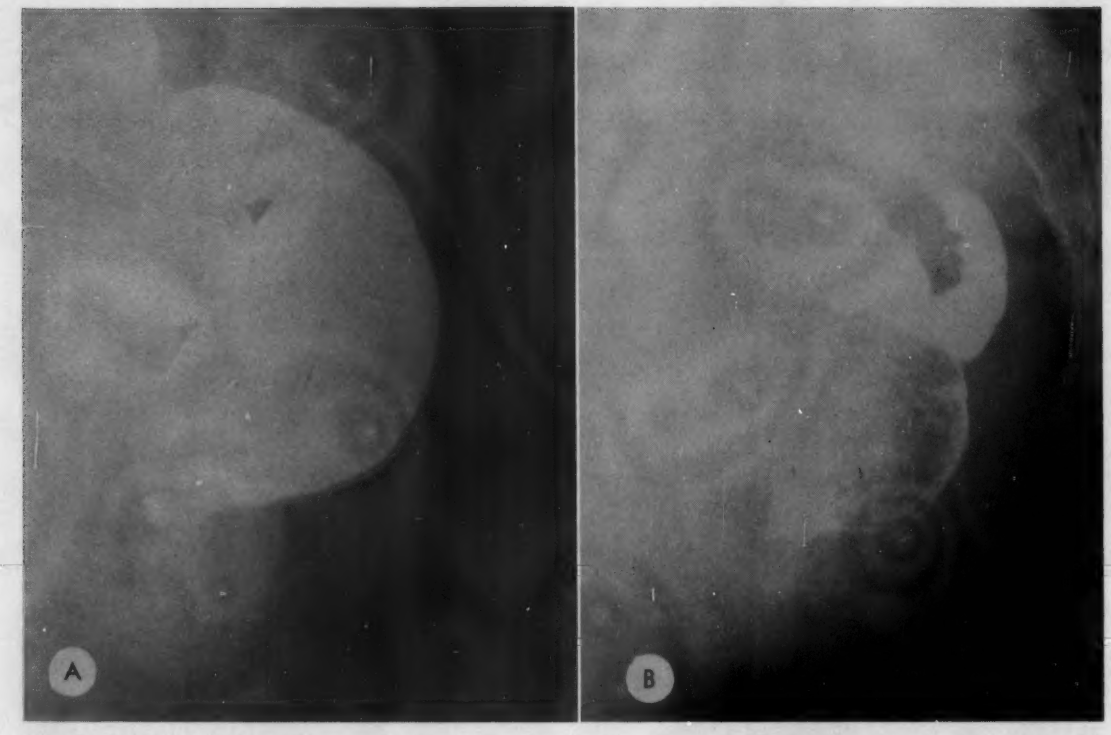

FIG. 4. (*A* and *B*) This patient complained of severe rectal tenesmus and constipation of six days' duration. Although the large scybalum seen on the postevacuation film was obvious fluoroscopically and on other roentgenograms, it is included here because it demonstrates so well how a large free-floating mass may be completely hidden by the barium in the filled rectum but is easily visible following evacuation.

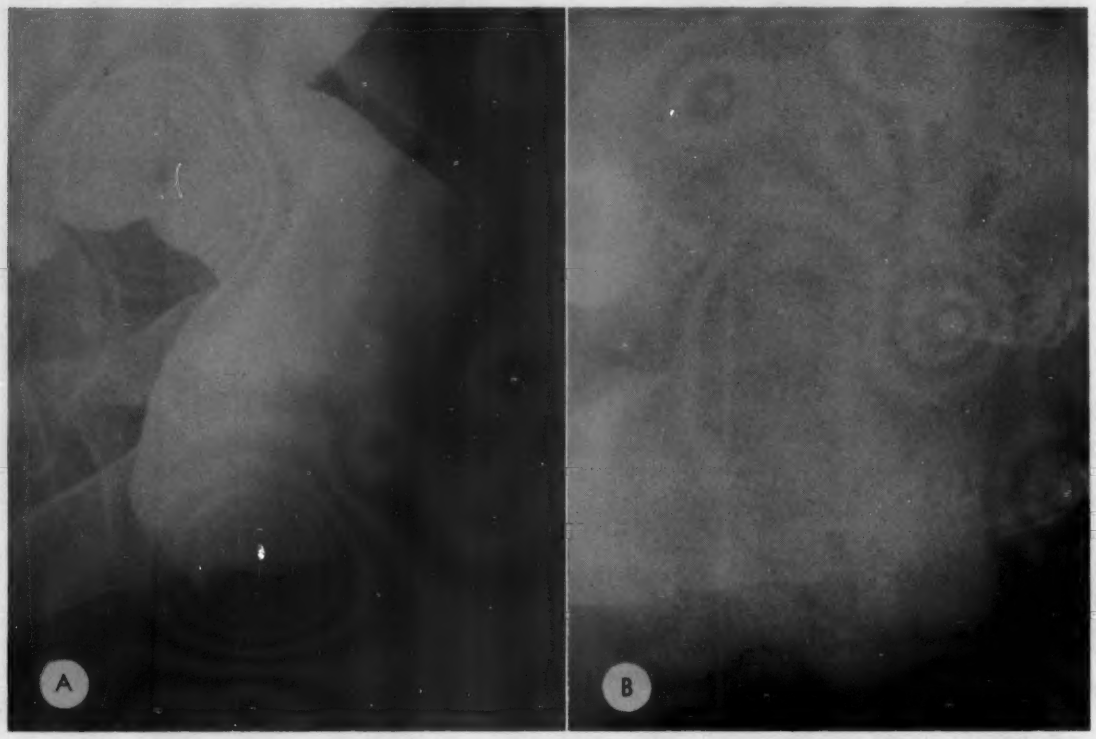

FIG. 5. (*A* and *B*) E.B., a forty-eight year old male, complained of increasing constipation, crampy lower abdominal pain and occasional melena. With the colon filled, no definite lesion could be seen in the capacious rectum although one might suspect some irregularity in the lower rectosigmoid; the remainder of the roentgenograms also revealed no lesion. Lateral roentgenogram following evacuation demonstrated a definite irregular filling defect in the upper rectum which proved to be due to adenocarcinoma.

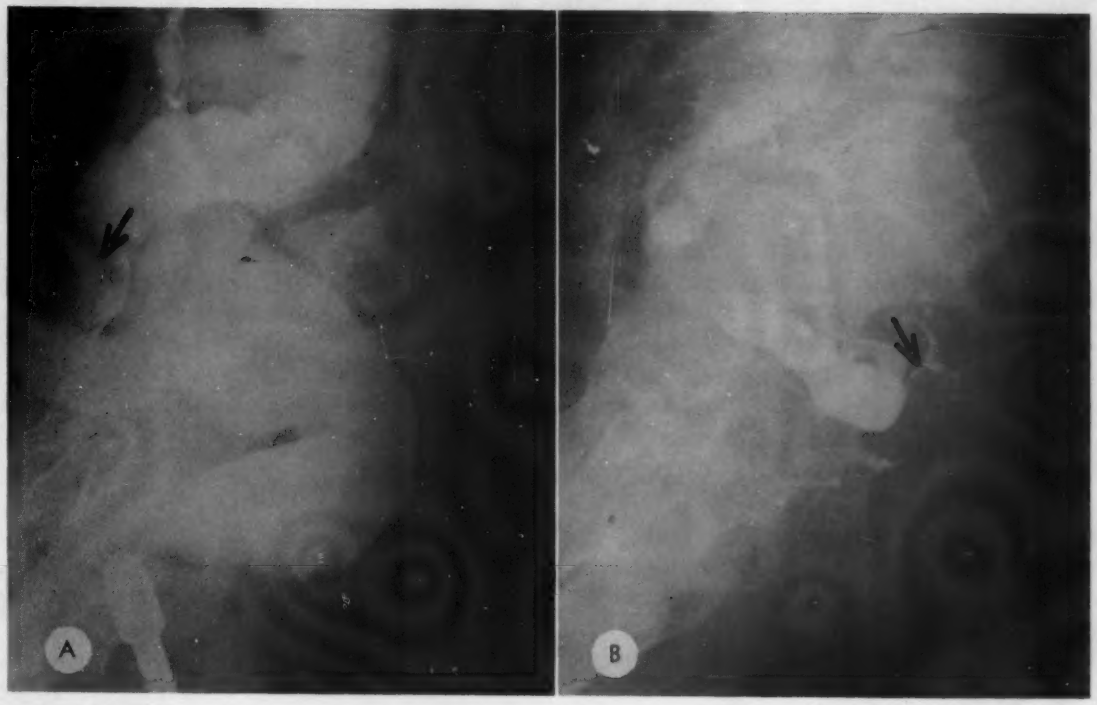

Fig. 6. (*A* and *B*) J.V., a twenty-seven year old housewife, had two episodes of massive bowel hemorrhage. Previous barium and air-contrast studies of the large bowel and two sigmoidoscopic examinations were reported as negative. Repeat examination revealed a highly mobile polyp easily seen on the postevacuation profile roentgenogram. At subsequent laparotomy, the polyp attached by a 10 cm. stalk was removed. The unusual mobility of this polyp explains its difficult visualization, both fluoroscopically and by sigmoidoscopy.

taining roentgenograms both before and after evacuation.

In an earlier publication from this department[5] it was shown that the physical factors required for the profile view render the method unreliable in detecting free floating polypi situated in the lower sigmoid colon and that air contrast study is essential to visualize this type of lesion. It was also demonstrated that the exposure dose delivered to the patient from its use is not excessive when proper technique is employed and when balanced against the benefits gained from its addition to the usual series of roentgenograms.

SUMMARY

A series of roentgenograms demonstrating the value of the routine lateral rectal projection is presented. The addition of this view to the usual series of roentgenograms provides valuable assistance in detecting lesions of the lower large bowel.

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INVOLVEMENT OF THE SACRUM BY RECURRENT CARCINOMA OF THE RECTUM*

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SACRAL involvement by recurrent rectal cancer has not been emphasized previously. It has been assumed to be too rare a condition to require a roentgenologic examination of the sacrum in patients with carcinoma of the rectum.[6] However, intense low back or sacrosciatic pain may result from involvement of the sacrum and sacral plexus by tumor.[1] Thus, persistent pain may be the earliest indication of recurrent malignancy, and may be followed by roentgenographically demonstrable changes in the sacrum. The purpose of this paper is to present a study of a group of patients with involvement of the sacrum by recurrent carcinoma of the rectum.

Records were examined of 125 patients who had had resection of rectal carcinoma and later developed persistent pain in the low back. Forty of the 125 had roentgenographic examinations of the sacrum (roentgenograms were reviewed only for patients with serial examinations or on whom roentgenograms had been taken within six months of death); 32 have died and 8 are living. Of the remainder of the 125 patients practically all are dead, although several could not be traced. The average length of life after onset of pain was about eighteen months; a few patients lived five years or longer.

Involvement of the sacrum by recurrent rectal carcinoma was noted in 26 of the 40 cases. One of these (No. 8) was diagnosed at necropsy as no roentgenographic examination of the sacrum had been made for three years prior to death. Three additional cases showed involvement of the coccyx, 2 with microscopic confirmation of carcinoma, but without demonstrable roentgenographic changes in the sacrum.

This group of 26 patients with roentgenographically detectable sacral involvement by recurrent rectal carcinoma was selected for this study and consisted of 15 males and 11 females. Excision of the rectum (15 abdominal-perineal, 1 abdominal-perineal proctectomy, 1 abdominal-perineal "pull-thru," 8 anterior resections and 1 posterior resection) had been performed in 11 different hospitals for cure of carcinoma. Nineteen of the 26 cases are dead; one is presumed to be dead and 6 are living. Four of the living patients appear to be in a terminal stage of recurrent malignancy. Detailed findings in the 26 patients are presented in Tables I and II.

OBSERVATIONS

Pain. Persistent pain, generally low back or sacral in location, was experienced by all 26 patients. A sudden onset occurred in 4 patients. In 15, pain radiated to the thighs or legs. The pain was constant, usually unrelated to change in position, unrelieved by rest, and was not worse at night. It became progressively more severe, reaching such intensity that large amounts of narcotics were required for partial relief. Twenty-four of the 26 patients were addicted to narcotics before sacral involvement was diagnosed.

Neurologic examinations initially were generally normal. Lumbar myelography was carried out in 3 patients. In one patient, intervertebral exploration of the L-5—S-1 space was performed, but no protruded disk was found.

Psychiatric consultation to evaluate the functional component of the intractable pain was obtained in 4 patients, since no organic basis could be established initially. Later, sacral changes became evident on roentgenograms, thus explaining the severe

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pain. In 3 of the 4 patients, the sacral abnormality remained the only objective evidence of recurrent tumor.

Radiation therapy was administered in 18 cases (Table I and II) in an attempt to relieve the pain. Partial relief was usually obtained, but in most cases the pain recurred rapidly. One patient was kept fairly comfortable for several years with multiple short courses of radiation therapy.

The remainder of the 125 patients whose records were examined presented findings generally similar to the detailed observations recorded in the group of 26 cases. The pain patterns were the same, initial neurologic examinations were usually normal and palliative radiation therapy afforded temporary relief in most patients.

Roentgen Findings. Roentgenograms of the sacrum (frontal and lateral projections) were reviewed in 21 patients who had had serial examinations; in one patient with a single examination, confirmation of sacral involvement was obtained at surgical exploration. The roentgenograms had been discarded in 3 cases. Two of these had microscopic confirmation of carcinoma in the sacrum after roentgenographic demonstration of sacral involvement; in another case (No. 5), osteoblastic changes were reported on two roentgen examinations. There were no recent roentgenograms for the case discovered at necropsy.

Sacral involvement by recurrent tumor consisted of osteolytic changes in 17, osteoblastic changes in 6 and a combination of destructive and productive changes in 2. A mass associated with sacral destruction was identified roentgenographically in the one patient in whom vaginorectal palpation had revealed a presacral tumor. Rectal palpation in 2 males, vaginorectal palpation in 4 females and vaginal palpation alone in 6 females failed to detect a mass.

Osteolytic changes in the 17 cases appeared as a loss of the anterior cortical line in 14 and as destruction of an inferior border in 2; an additional case (No. 6) was reported as showing "destruction of the right sacrum." In 3 of the 14 in which loss of the cortex was identified on the lateral projections, destruction of the sacrum was apparent in the frontal views (Fig. 1). Two cases of destruction of the sacrum not evident on the lateral roentgenograms were identified on the frontal projections.

Osteoblastic involvement in 6 cases was identified in both frontal and lateral projections (Fig. 2). However, the changes were usually more apparent on the lateral roentgenograms except where massive involvement had occurred. In 3 patients progression of the osteoblastic changes in the sacrum was noted on serial roentgenograms.

The cortex in the 6 cases with osteoblastic involvement of the sacrum appeared preserved in one case (Fig. 2), markedly thickened in another (Fig. 3) and in 3 was indistinguishable from the productive changes in the vertebral body. One of the 3 cases showed marked new bone formation in the presacral region (Fig. 4). Roentgenograms were not reviewed of one patient with osteoblastic changes.

The third segment of the sacrum, in the "hollow," was frequently the first demonstrable site of involvement by recurrent tumor; this was observed roentgenographically and at surgical exploration. Roentgenographic demonstration of involvement of either the second or fourth sacral segments after identification of an abnormality in S-3 was noted in 5 patients. In the cases in which the malignancy involved more than one sacral segment, the portions affected were contiguous. The sacral foramina appeared intact in all of the patients.

Laminagraphic examinations demonstrated involvement of the sacrum in 5 recently diagnosed cases. However, no sacral changes were detected by this method which were not evident on conventional roentgenograms.

The sacrum in 3 patients was visualized in the lateral projection of the barium-filled colon, in which a primary tumor was demonstrated in the distal large bowel. Two of the 3 later showed involvement of the sa-

TABLE I

PATIENTS WITH SACRAL INVOLVEMENT BY RECTAL CARCINOMA WHO HAVE DIED

No. Age Sex	Operation	Pain	Roentgen Findings (frontal and lateral projections unless stated differently)	Date of Death	Confirmation of Sacral Involvement by Recurrent Carcinoma
1. 51 F (R.B.)	9/20/40 Abdominal-perineal resection	Low back, radiating down each leg, March, 1946 (Cordotomy: 6/10/48)	4/ 7/47 Normal *Radiation therapy* 6/10/47 Destruction of anterior cortical margin of S-1 1/14/48 Bone repair of S-1 5/ 1/48 No change 5/10/48 No change	12/ 4/48	
2. 67 F (A.B.)	2/11/52 Abdominal-perineal proctectomy	Sacral, December, 1952	1/20/53 Normal 3/ 7/53 Normal 8/27/53 Normal 4/ 5/54 Osteolytic and osteoblastic changes in S-3 through S-5 4/ 7/54 As above 4/19/54 As above	12/30/54	
3. 54 F (S.U.)	11/5/55 Anterior resection with end-to-end anastomosis 7/4/57 Surgical exploration for intestinal obstruction. Biopsy of peritoneum: Carcinoma	Low back, radiating down each thigh posteriorly, November, 1956	9/28/51 Normal 10/19/51 Normal (Fig. 2, *A* and *B*) 10/14/53 Normal 2/25/57 Osteoblastic involvement in S-2 5/24/57 No change *Radiation therapy* 7/27/57 Osteoblastic changes in S-1 and S-2 (Fig. 2, *C* and *D*) *Radiation therapy*	3/12/58	Necropsy: S-1 and S-2 "totally" replaced by carcinoma
4. 73 M (A.M.)	February, 1949 Abdominal-perineal resection	Low back, radiating down each leg, March 1950 (Cordotomy: 2/18/52)	*Radiation therapy* 9/18/51 Destruction of anterior cortical margin of S-1 *Radiation therapy* 2/11/52 No change	7/26/52	
5. 46 M (D.R.)	1/23/31 Abdominal-perineal resection	Perineal, radiating down each leg, December, 1931 (Cordotomy: 1/19/34)	*Radiation therapy* 1/18/34 Osteoblastic metastases in S-1 2/16/34 No change (Roentgenograms not reviewed)	3/15/34	
6. 34 F (A.S.)	4/9/32 Posterior resection 12/18/33 Partial resection of sacrum: Carcinoma in sacrum (Perineal sinus, persistent) 11/8/35 Perineal scar biopsy: Carcinoma	Right sacral, March, 1933 (Cordotomy: 5/24/34)	11/25/33 Destruction of right sacrum (Roentgenogram not reviewed)	9/19/37	Microscopic diagnosis: Carcinoma in sacrum
7. 68 M (A.R.)	November, 1948 Abdominal-perineal resection 11/15/50 Perineal dissection for recurrent carcinoma. Sacral biopsy: Carcinoma	Perineal, August, 1950	*Radiation therapy* 10/15/51 Destruction of anterior cortical margin of S-5	10/19/51	Microscopic diagnosis: Carcinoma in sacrum
8. 42 M (N.W.)	5/1/33 Abdominal-perineal resection 11/7/38 Cystoscopy and bladder wall biopsy: Carcinoma 4/24/39 Perineal scar biopsy: Carcinoma	Sacral, radiating down left leg, April, 1935	Roentgenograms taken in 1935 and 1936 have been discarded *Radiation therapy:* 1936, 1937	11/23/39	Necropsy: Sacrum completely replaced by carcinoma

TABLE I—(*Continued*)

No. Age Sex	Operation	Pain	Roentgen Findings (frontal and lateral projections unless stated differently)	Date of Death	Confirmation of Sacral Involvement by Recurrent Carcinoma
9. 68 M (J.P.)	1/18/45 Abdominal-perineal resection	Left hip, radiating into left thigh, August, 1949	1/17/50 Normal 6/24/50 Frontal: Destruction of inferior margin, left side (osteolytic metastases of left pubic bone) Lateral: Normal 7/19/50 No change *Radiation therapy* 4/19/51 Partial bone repair of destroyed areas	7/30/51	
10. 53 F (A.F.)	2/3/56 Abdominal-perineal resection	Low back, radiating down left thigh, July, 1957	1/31/56 Normal 11/18/57 Destruction of anterior cortical margins, S-1 and S-2 *Radiation therapy* 3/10/58 Partial bone repair of S-1 and S-2	6/ 3/58	
11. 46 M (J.M.)	3/2/48 Anterior resection with end-to-end anastomosis 3/12/49 Abdominal-perineal attempted resection. Tumor densely adherent to sacrum anteriorly at least as high as S-3. Radium needle implantation into recurrent rectal cancer. Ileal and bladder wall biopsy: Carcinoma (Persistent perineal sinus) 8/15/49 Biopsy of margin of perineal sinus: Carcinoma 7/29/50 Surgical exploration for intestinal obstruction	Low back, September, 1949	3/ 8/49 Normal 3/14/49 Normal (Fig. 3*A*) 4/16/49 Normal 7/19/50 Frontal: Osteoblastic changes in midsacrum 7/22/50 As above 8/24/50 Periosteal new bone formation on anterior cortical margin of S-2 and S-3, at level of primary and recurrent rectal carcinoma (Fig. 3*B*) *Radiation therapy*	11/ 1/50	Surgical exploration: Tumor densely adherent to sacrum
12. 60 M (H.W.)	4/16/54 Abdominal-perineal resection 12/6/56 Drainage of deep perineal abscess 12/21/56 Exploratory laparotomy for perforation of small intestine. Drainage of pelvic abscess. Biopsy of ileum: Carcinoma	Perineal, September, 1956	11/30/56 Normal 12/14/56 Normal 12/17/56 Normal 12/30/56 Normal *Radiation therapy* 4/16/57 Normal 9/19/57 Destruction of anterior cortical margin of S-2 through S-5 9/22/57 No change 9/24/57 No change 1/28/58 No change	4/30/58	Necropsy: Presacral abscess. Osteomyelitis of sacrum. Carcinoma was not found in one block of tissue from sacrum
13. 42 F (H.W.)	1/19/48 Anterior resection with end-to-end anastomosis 9/17/48 Resection of rectum and sigmoid. Tumor markedly adherent to "hollow" of sacrum. Biopsy of peritoneum: Carcinoma	Perineal, August, 1948 (Prefrontal lobotomy: 2/19/49)	1/24/49 Destruction of anterior cortical margin of S-3	2/24/49	Surgical exploration: Tumor densely adherent to sacrum
14. 55 M (C.G.)	April, 1950 Abdominal-perineal resection 6/19/52 Exploratory laparotomy. Extensive tumor in pelvic cavity. Ileal and peritoneal biopsies: Carcinoma (Sinus from midline operative scar began draining April, 1953)	Right sacral, going into right buttock and radiating down posterior thigh (onset unknown)	7/11/50 Normal 4/22/52 Normal *Radiation therapy* 7/14/52 Frontal: Destruction of inferior portion on right side Lateral: Destruction of anterior cortex S-3 through S-5 9/11/52 No change 3/ 2/53 Frontal: No change	6/18/53	

TABLE I—(*Continued*)

No. Age Sex	Operation	Pain	Roentgen Findings (frontal and lateral projections unless stated differently)	Date of Death	Confirmation of Sacral Involvement by Recurrent Carcinoma
15. 42 F (T.R.)	7/9/51 Anterior resection, end-to-end anastomosis 9/5/52 Abdominal-perineal resection. Excision, distal half of sacrum: Carcinoma 5/11/53 Nephrostomy, left 6/ 3/53 Nephrostomy, right 8/28/53 Aspiration biopsy of sacrum: Carcinoma	Right coccygeal, May, 1952 (Cordotomy: 10/1/53)	6/ 3/52 Frontal: Destruction of inferior margin, right Lateral: Normal 7/ 3/52 No change *Radiation therapy*	3/ 5/54	Microscopic diagnosis: Carcinoma in sacrum
16. 51 M (P.G.)	December, 1951 Abdominal-perineal "pull-thru" 9/18/53 Abdominal-perineal resection for recurrent cancer in distal large intestine 2/25/55 Exploratory laparotomy. Pelvic floor solid with tumor. Perineal biopsy: Carcinoma	Coccygeal, radiating down right thigh anteriorly to knee, April, 1954	9/22/54 Destruction of anterior cortex, S-4 and S-5 2/23/55 Destruction of anterior cortical margin, S-3 through S-5 12/12/55 Destruction of anterior cortex of S-2 through S-5 *Radiation therapy*	6/ 3/56	Necropsy: Sacrum replaced by carcinoma
17. 58 M (W.P.)	September, 1953 Abdominal-perineal resection January, 1957 Excision of coccyx: No carcinoma	Perineal and left sacral, September, 1955 (Cordotomy: September, 1958)	8/20/57 Normal 8/23/57 Normal (Fig. 1, *A* and *B*) 2/24/58 Frontal: Destruction of inferior margin on left side Lateral: Destruction of anterior cortex, S-3 through S-5 3/ 4/58 As above (Fig. 1, *C* and *D*) *Radiation therapy* 6/ 5/58 Bone repair of destroyed segments (Fig. 1, *E* and *F*) 10/ 1/58 Destruction of anterior cortex of S-2 (Fig. 1*G*)	12/15/58	
18. 53 M (A.G.)	12/15/52 Abdominal-perineal resection	Coccygeal, radiating down right leg, December, 1952 (Rhizotomies: 1/31/55, 3/1/55)	6/ 9/52 Normal 11/25/53 Normal 1/14/54 Normal 5/28/54 Normal 5/ 9/55 Osteolytic and osteoblastic changes, S-2 through S-5 (metastases in right ischium) 5/26/55 As above	6/12/55	
19. 53 F (L.J.)	1/22/47 Abdominal-perineal resection 9/9/53 Excision of nodule in perineal scar: Carcinoma	Low back and coccygeal, September, 1949	7/ 8/47 Normal 12/30/52 Destruction of anterior cortex, S-2 through S-5. Extensive demineralization 9/ 8/53 No change *Radiation therapy*	11/18/55	
20. 44 M	11/13/33 Abdominal-perineal resection with excision of coccyx and last segment of sacrum 12/8/34 Excision of lower end of sacrum: Carcinoma	Unknown	12/ 5/34 Osteoblastic involvement in S-4 (Roentgenograms not reviewed)	Presumed dead	Microscopic diagnosis: Carcinoma in sacrum

TABLE II

LIVING PATIENTS WITH SACRAL INVOLVEMENT BY RECURRENT RECTAL CARCINOMA

No. Age Sex	Operation	Pain	Roentgen Findings	Confirmation of Sacral Involvement by Recurrent Carcinoma
21. 71 F (C.P.)	3/28/55 Anterior resection with end-to-end anastomosis (3/7/59 Vaginorectal palpation: Large hard mass in "hollow" of sacrum)	Low back and perineal, radiating down right leg posteriorly, December, 1958	3/ 7/59, 3/10/59 Destruction of anterior cortex of S-3 *Radiation therapy*	
22. 55 F (M.W.)*	4/1/52 Anterior resection with end-to-end anastomosis 2/26/59 Exploratory laparotomy. Aspiration biopsy of thrombosed vena cava: Carcinoma	Low back, radiating down left leg posteriorly, April, 1957	3/24/52 Normal (Fig. 5*A*) 3/31/52 Normal 9/ 5/58, 9/18/58, 9/22/58 Destruction of anterior cortex of transitional S-1 at level of primary rectal cancer (Fig. 5, *B–D*) *Radiation therapy* 11/12/58 No change 1/15/59 No change 2/ 6/59 Minimal collapse, S-1	
23. 67 M (L.S.)*	4/28/50 Abdominal-perineal resection 3/27/59 Cystoscopy and bladder wall biopsy: Carcinoma	Perineal and right hip, radiating down posterior thigh and leg, May, 1954	5/17/54 Normal (Fig. 4, *A* and *B*) 1/ 9/58, 2/28/58 Osteoblastic involvement, S-3 through S-5 *Radiation therapy* 8/ 1/58, 8/ 8/58, 9/23/58, 9/29/58, 10/ 9/58 Osteoblastic changes, S-2 through S-5 (Fig. 4, *C* and *D*) *Radiation therapy* 3/26/59 Involvement of entire sacrum and coccyx	
24. 57 M (A.S.)*	3/18/55 Abdominal-perineal resection	Perineal, radiating down right thigh posteriorly, March, 1955 (pain began several days after operation) (Cordotomy: 7/27/56)	7/26/56 Normal 12/22/56 Normal 8/15/58, 8/29/58 Osteoblastic changes, S-3 through first segment of coccyx 1/30/59 Involvement, S-2 through coccyx	
25. 50 F (L.R.)*	November, 1952 Abdominal-perineal resection April, 1958 Intervertebral exploration, L-5/S-1 (no protruded disk) 3/11/59 Exploratory laparotomy. Aspiration biopsy of sacrum: Carcinoma	Perineal, radiating down right leg, January, 1958 (Cordotomy: May, 1958)	*Radiation therapy* 1/31/59, 2/ 5/59 Frontal: Destruction of right hemisacrum superiorly Lateral: Destruction of anterior cortex, S-1 and S-2 *Radiation therapy* 2/24/59 No change	Microscopic diagnosis: Carcinoma of sacrum
26. 63 M (M.S.)	1/27/58 Anterior resection with end-to-end anastomosis 6/4/58 Right frontal lobectomy for meningioma	Low back (onset unknown)	8/26/58, 9/ 9/58, 12/ 5/58, 12/10/58 Destruction of anterior cortical margin, S-1 and S-2	

* In terminal stage of recurrent cancer.

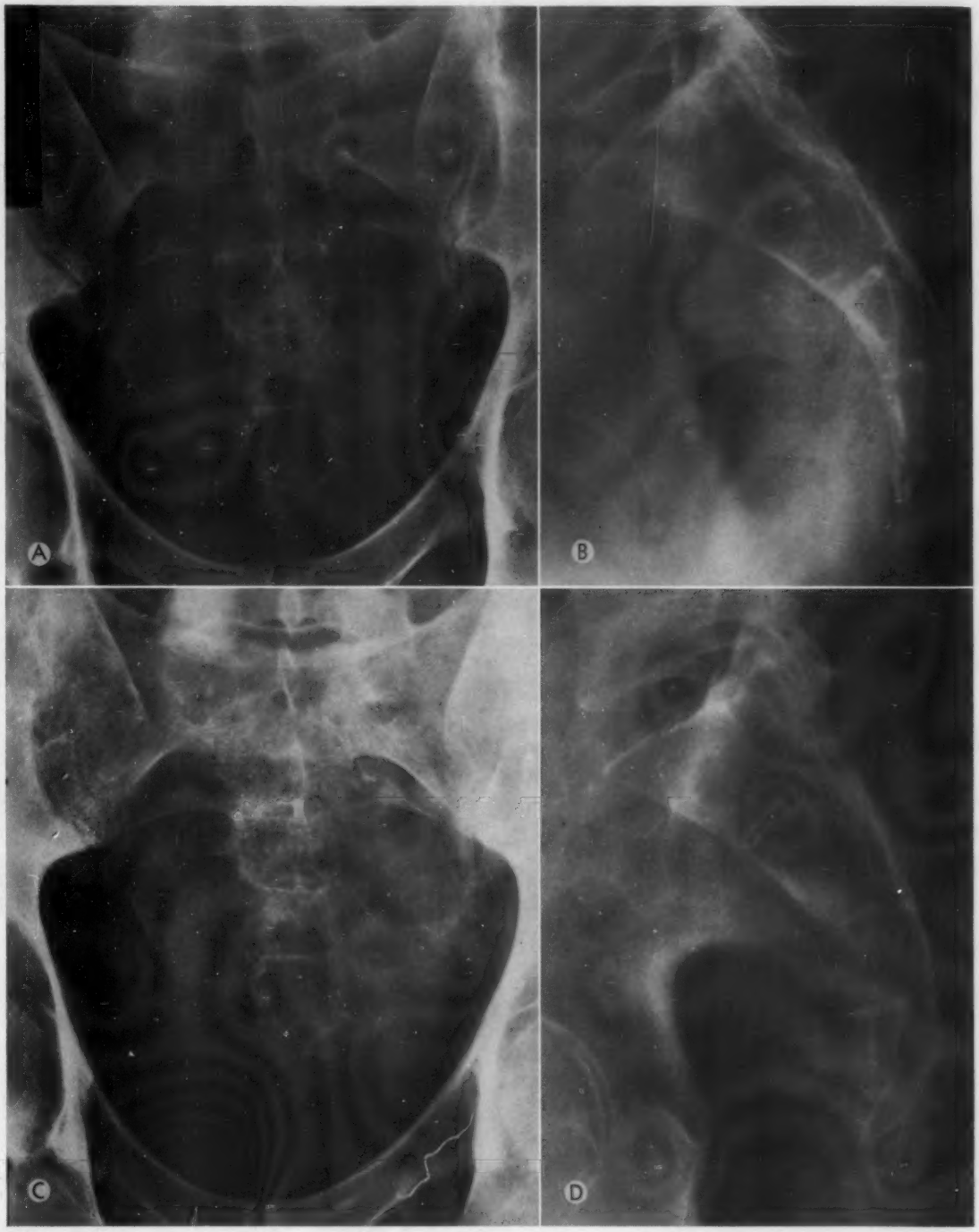

Fig. 1. (Case No. 17). Osteolytic involvement of the sacrum in a fifty-eight year old man who had had resection of rectal cancer in September, 1953. (*A* and *B*) August 23, 1957: Normal sacrum. The coccyx has been excised. (*C*) March 4, 1958: Destruction of left inferior margin. (*D*) March 4, 1958: Loss of anterior cortex of S-3.

FIG. 1. (*E* and *F*) June 5, 1958: Bone repair of destroyed segment two months after radiation therapy. (*G*) October 1, 1958: S-2 is now destroyed.

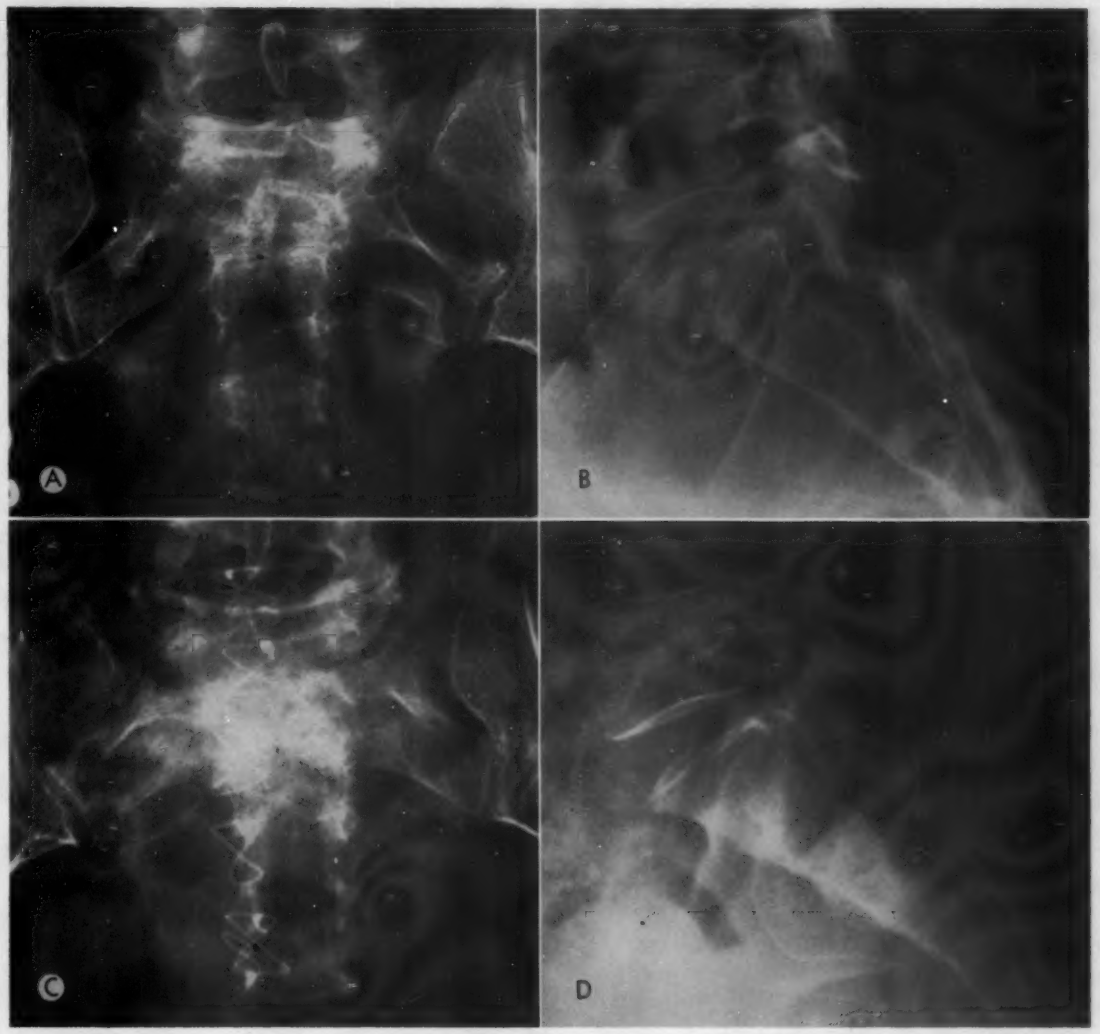

Fig. 2. (Case No. 3). Carcinoma in sacrum confirmed by necropsy in a fifty-four year old woman with rectal cancer excised two years previously. (*A* and *B*) October 19, 1951: Normal sacrum. (*C* and *D*) July 27, 1957: Osteoblastic involvement of the body of S-1 and S-2 with preservation of anterior cortex.

crum at the level of the rectal cancer (Fig. 5).

Radiation therapy was given to 13 of the 17 patients with osteolytic changes in the sacrum. Four showed bone repair of the destroyed segments of the sacrum; 2 were unchanged and 7 were not followed roentgenographically after radiation therapy.

Pathologic Findings. Microscopic confirmation of carcinoma in the sacrum was obtained in 7 patients after the sacral changes were demonstrated roentgenographically. In 4 carcinoma was found after surgical excision of a portion of the sacrum, in one patient after aspiration biopsy and in 2 at necropsy. In one patient, examined post mortem, carcinoma involved the entire sacrum; in the other, the first and second segments were "totally" replaced by tumor.

In one case (No. 8), necropsy demonstrated replacement of the sacrum by cancer (no roentgenograms). Surgical exploration in 2 cases disclosed tumors densely adherent to the sacrum; biopsy specimens were not obtained.

Necropsy on an additional case (No. 12)

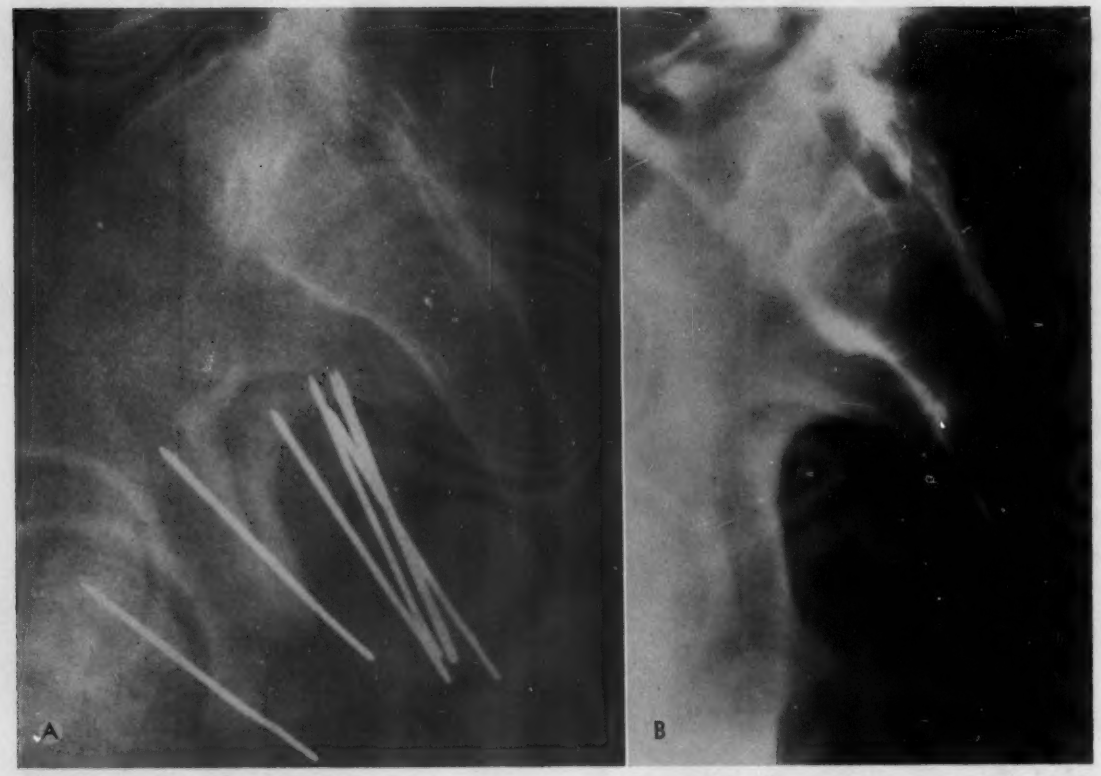

FIG. 3. (Case No. 11). Periosteal new bone formation in a forty-six year old man in whom surgical exploration revealed a tumor densely adherent to midsacrum. Excision of rectal carcinoma in March, 1948. (*A*) March 14, 1949: Normal sacrum. Radium needles have been implanted into recurrent rectal cancer. (*B*) August 24, 1950: Cortical thickening of anterior margin of S-2 and S-3.

revealed a presacral abscess and osteomyelitis of the sacrum; roentgenograms showed extensive destruction of the anterior cortex from S-2 through S-5. Carcinoma was not found in one block of tissue taken from the sacrum, but extensive carcinoma was present in the pelvic cavity and right ileum. Multiple sections from each sacral segment might conceivably have disclosed carcinoma in addition to the osteomyelitis of the sacrum.

Involvement of the sacrum demonstrated roentgenographically was the earliest evidence of recurrent rectal cancer in 24 of the group of 26 cases. In the remaining 2 patients, metastases in the bony pelvis were apparent at the time of detection of sacral involvement. Later, carcinoma in structures other than the sacrum was identified by microscopic examination in 7 of the 24 patients (in addition to the 8 cases with histologically proved cancer in the sacrum). In another patient, pulmonary metastases became evident roentgenographically six months after recognition of sacral destruction by recurrent tumor.

Three patients had draining sinuses from the operative sites. Carcinoma was microscopically confirmed by biopsy specimens from the sacrum in one patient, from a margin of the perineal sinus tract in another and from the peritoneum in the remaining patient.

DIFFERENTIAL DIAGNOSIS

Recurrence of rectal cancer is usually difficult to recognize after excision of the rectum, particularly after abdominal-perineal resections in men. In this group palpation of the perineum and perineal scar gave little information, particularly in the excessively fibrotic scars. Proctoscopy (in 8

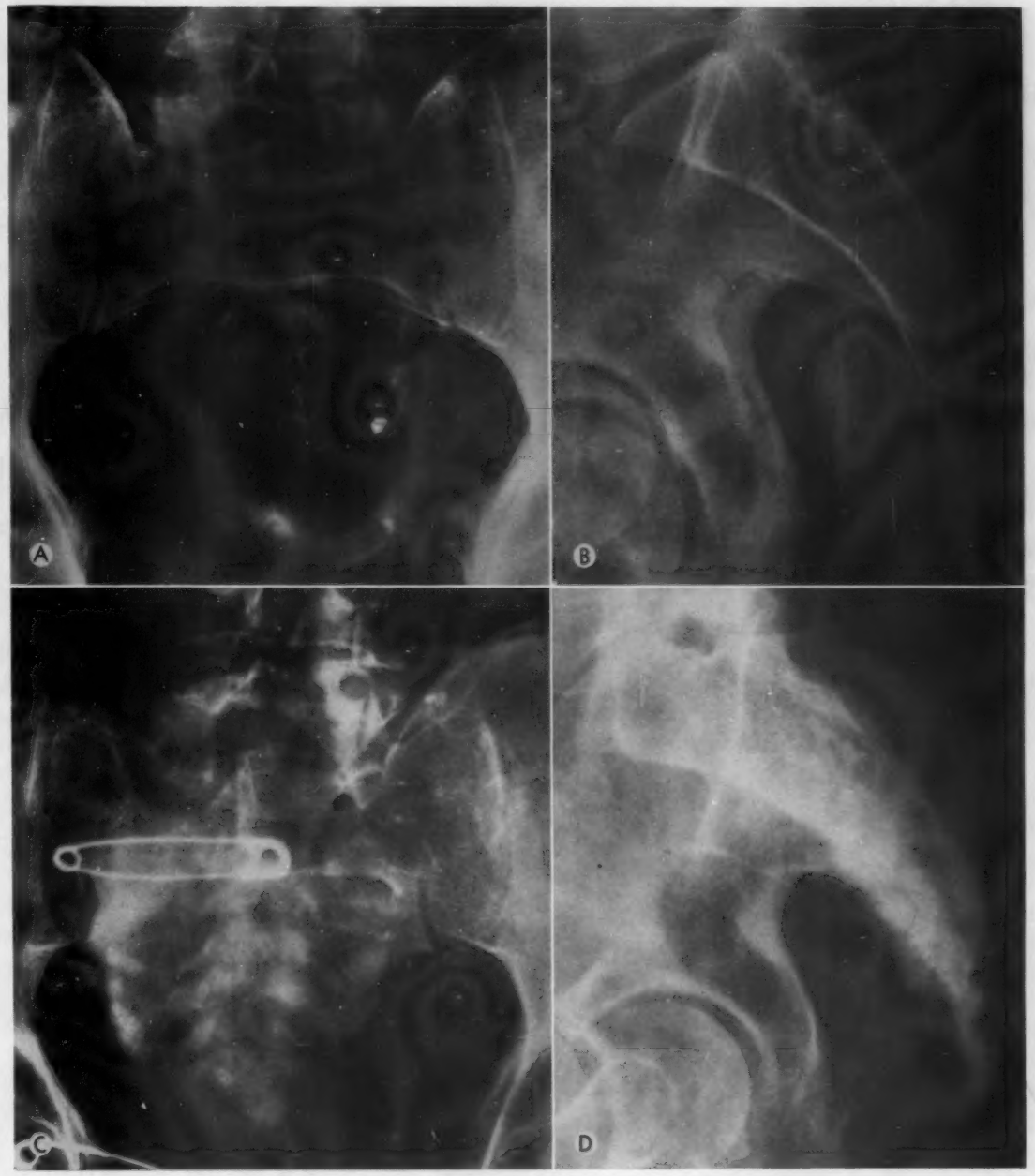

Fig. 4. (Case No. 23). Osteoblastic involvement of sacrum in a sixty-seven year old man with rectal cancer resected eight years previously. (*A* and *B*) May 17, 1954: Normal sacrum. (*C* and *D*) August 1, 1958: Productive changes in S-2 through S-5 with marked formation of new bone in presacral area.

patients in whom sphincter-saving operations had been performed) was of no value in determining recurrence. Palpation (vaginal and/or rectal) in 13 patients revealed a mass in only one instance.

Neurologic examination early in the course of tumor involvement of the sacrum with impingement on the sacral nerves or lumbosacral plexus is usually normal, particularly in older people. Localization of a lesion by the nerve deficits may be possible later as the tumor progresses. However, a differential diagnosis must be made between carcinoma in the sacrum affecting

FIG. 5. (Case No. 22). Destruction of S-1 (transitional) in a fifty-five year old woman with excision of rectal carcinoma six and one-half years previously. (*A*) March 24, 1952: Barium-filled distal large bowel shows a long segment of narrowed large intestine at level of intact S-1. (*B*) September 5, 1958: Loss of anterior cortex of S-1 adjacent to anastomosis. (*C* and *D*) September 18, 1958: The destruction of S-1 is evident in the lateral view but not in the frontal view.

the nerves and other conditions causing sciatic pain,[9] particularly protruded intervertebral disks (15 of the 26 patients with sacral involvement by recurrent carcinoma had radiation of pain down the thighs). Special procedures, such as myelography, may be required to aid in further localization and identification of the nature of the abnormality producing the nerve deficits.

Thus, roentgenographic demonstration of sacral involvement in a patient with resected rectal cancer contributes signifi-

cantly in establishing a diagnosis of recurrence of malignancy. However, sacral changes are not exclusively characteristic of recurrent rectal carcinoma; the sacrum may also be involved by osteomyelitis[4] and by a wide variety of tumors, both primary and metastatic.[5]

Osteomyelitis must be considered when either osteolytic or osteoblastic changes are noted in the sacrum. The anterior cortex of the sacrum may be destroyed by a presacral abscess with subsequent development of osteomyelitis (No. 12), producing an appearance roentgenographically identical with that observed in tumor involvement. However, osteomyelitis generally causes a productive change in the surrounding bone; it usually begins in or near the sacroiliac joints.[5] Occasionally, osteomyelitis may be associated with carcinoma in the sacrum, since breakdown of recurrent tumor in the intestinal tract may result in a presacral abscess with development of osteomyelitis later. These two conditions cannot be distinguished with certainty roentgenographically.

Primary tumors of the sacrum may arise within the sacral canal or from the body of the sacrum. Erosion of the sacral canal, which occurs in tumors arising within this structure, was not seen in our group of 26 cases. The sacral foramina were also intact, thus excluding neurofibroma. Tumors arising from the body of the sacrum, such as chordomas, usually cause expansion of the sacrum, particularly in the anteroposterior diameter. In cases of sacral involvement by recurrent rectal cancer, expansion of the sacrum was not observed.

Metastatic tumors of the osteolytic type frequently involve multiple areas of the sacrum and other portions of the skeleton, and do not usually destroy the anterior cortex of a sacral segment. Osteoblastic metastases from prostatic malignancy do not produce the progressive segmental involvement observed in recurrent rectal cancer. They usually involve multiple areas in the sacrum, pelvis and lumbar spine instead of localizing mainly to one side of the sacrum. Such metastases do not produce the changes in the cortex observed in cases of recurrent rectal carcinoma.

COMMENT

Sacral involvement by carcinoma of the rectum has been observed previously. Bacon and Gilbert[3] reported on skeletal involvement in a series of 318 patients with malignancy of the anus, rectum and sigmoid colon. All of the 318 patients had metastases to one or more organs as evidenced by biopsy, surgical exploration, roentgenograms or necropsy. Sacral involvement was noted in 22 instances. In 18 cases the coccyx was involved, but they do not state the number with co-existent tumor in the sacrum. Aufses[2] reported one case of rectal carcinoma involving the sacrum at necropsy and another with roentgen changes. Gross[7] observed destruction of the sacrum in the frontal projection in a case of silent rectal carcinoma; necropsy confirmed the presence of carcinoma in the sacrum.

The importance of recognition of sacral involvement by recurrent rectal cancer needs emphasis. Roentgenographic demonstration of sacral changes contributes significantly to the detection of recurrence of malignancy of the rectum. Proper management of this group of patients is easier when a definite diagnosis of recurrent cancer is made before placing a patient on large doses of narcotics or resorting to palliative procedures such as radiation therapy or neurosurgery for relief of pain.

Persistent pain in the low back in any patient with resected rectal cancer is *presumptive* evidence of recurrent tumor. A long interval may elapse from the time of excision of the carcinoma of the rectum until the pain appears; 5 in this group of 26 patients lived over five years before developing pain from sacral involvement.

The earliest indication of a recurrence of rectal malignancy may be constant low back pain. Twenty-four of our patients

suffered persistent pain which preceded by several months other evidence of recurrent tumor.

The sacrum should be examined roentgenographically at the time of initial diagnosis of rectal cancer. The lateral projection of the barium-filled rectum should include the sacrum as well. The level of the primary tumor in relationship to the sacrum is thus established and on subsequent examinations of the sacrum, after pain develops, particular attention may be directed to this area. In 2 of the 3 patients on whom lateral roentgenograms had been taken initially, the involvement of the sacrum occurred at the level of the primary rectal carcinoma.

A lateral roentgenogram of the sacrum taken at the time of diagnosis of rectal cancer also furnishes a normal view with which to compare subsequent roentgenograms. Destruction of the sacrum may be difficult to identify without this initial examination to confirm a change. It is particularly important to obtain a direct lateral projection; slight rotation of the sacrum may produce distortion of the anterior cortical line which simulates destruction.

The importance of the lateral projection is further demonstrated by the fact that in 14 patients there was destruction of the anterior cortex apparent in the lateral view, but only 3 showed an abnormality in the frontal projection as well. Moreover, in only 2 patients was destruction noted on the frontal roentgenogram and not on the lateral roentgenogram. Osteoblastic changes in the sacrum were also generally more evident on the lateral than on the frontal roentgenograms. The limited value of the examination of the sacrum in the frontal projection is also emphasized by the observation that destruction which had occurred was not visualized in this view (in several cases almost the entire anterior cortex of the sacrum was destroyed without a demonstrable abnormality on the frontal roentgenogram).

The difficulty of recognizing early bone changes in the sacrum has been pointed out by Paulson.[8] Partial obscuration of the sacrum in frontal projections by gas and fecal matter in the intestinal tract makes proper interpretation very difficult. Destruction of the anterior cortex, often in the midline, of the upper and midsacrum is superimposed on a thicker portion of underlying intact bone. Involvement of an inferior margin is easier to identify; in 5 cases which had such involvement, the abnormality was noted in the frontal projection. The lower segments are normally so variable in appearance, possibly in relation to mineral content, that involvement by tumor is difficult to identify.

The method by which carcinoma involves the sacrum is unknown. Extension by continuity of tissue, spread by lymphatic drainage, or implantation of malignant cells in the periosteum of the sacrum at the time of excision of the rectum are all possibilities.

The periosteum of the sacrum appears to be a fairly effective barrier to the passage of cancer into the body of the sacrum. The localized cortical thickening (No. 11) at the site of adherence of tumor to the sacrum is a protective reaction by the periosteum to prevent spread of carcinoma into the sacral vertebrae. The protection afforded by the periosteum may account for the long interval which elapsed in 5 patients from the time of excision of rectal cancer until sacral involvement became evident.

Recurrent rectal carcinoma may also involve the coccyx.[3] Roentgenographic demonstration of this involvement, in conjunction with changes in the sacrum, may be obtained occasionally (No. 23, 24); however, microscopic evidence of carcinoma in the coccyx is the usual method by which the diagnosis is established.

The incidence of involvement of the sacrum by carcinoma of the rectum has not been determined by necropsy. Sections of the sacral vertebrae are usually not obtained at postmortem examinations unless some gross abnormality in the segments is

recognized. Thus, it is quite probable that many cases of carcinoma in the sacrum are not diagnosed. Serial roentgenographic examinations and a careful search at necropsy should reveal the incidence of sacral involvement by carcinoma of the rectum.

SUMMARY

1. Twenty-six cases of sacral involvement by recurrent carcinoma of the rectum are reported.

2. Persistent pain in the low back after excision of rectal cancer is *presumptive* evidence of recurrent tumor involving the sacrum.

3. Destruction of the anterior cortex was the most frequent roentgenographic finding.

4. The importance of the lateral projection is emphasized.

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REMOTE CONTROL FOR CINEFILM PROJECTOR*

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FOR satisfactory analysis and interpretation of roentgen-ray motion pictures, the projector should be equipped to allow the radiologist to easily alter projection speed, change direction or switch to single frame viewing. In the presentation of such studies to a group, it is highly desirable to have these features available at a distance from the projector, *i.e.*, remote control. This paper describes a simple and economic alteration of an already available projector to fulfill this purpose (Fig. 3).

The Weinberg-Watson modification of the Kodak Analyst II projector available in our radiology department provided manual speed and film direction control on the projector and a hand-held instrument to control film direction and single framing. The latter device was used by us as a basis for developing a detachable system of remote control of the projector with switches for film speed, direction and single framing. The modifications were carried out in the following manner.

To accomplish mechanical control of the projector speed rheostat, its shaft was connected by means of a worm and gear drive to a small D.C. motor (Fig. 5, A and B) equipped with a suitable power supply (Fig. 4) and wired to a switch in the hand control box. This allowed remote operation of the shaft and thus instantaneous control of the projector speed (Fig. 1).

It was found, however, that this system operated too rapidly to permit sufficiently small changes in the rate of projection. Therefore, a variable resistance was installed in series with one pole of the motor (Fig. 5, C). This could be adjusted to provide the degree of control desired by the operator. However, in order to rapidly stop projection for single frame viewing of a particular portion of the film, a faster means of speed control was needed. This was obtained by bypassing the variable resistance through a push-button switch installed in the remote control box (Fig. 2, C).

All connections to the hand control box, including those originally present, were made with a 12 conductor cable and socket (Fig. 2, F and G). With the remote control cable detached, a shorting plug inserted into the socket allowed projector operation by means of the original controls located on the projector housing (Fig. 6).

The various switches were placed in the control box in positions so as to facilitate single handed operation. The shape of each switch was designed for tactile identification in the dark (Fig. 2).

By the use of this device the operator may, at a distance from the projector, change the projection speed slowly or quickly; he can change film direction or stop it almost instantaneously for single framing.

In practice, this has been found to greatly facilitate the repetitive study of cineroentgenographic films, so necessary for their proper interpretation. Furthermore, it has proved particularly useful for presentation of films before large groups at conferences and medical meetings.

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(*Figures 1–6 are shown on following pages.*)

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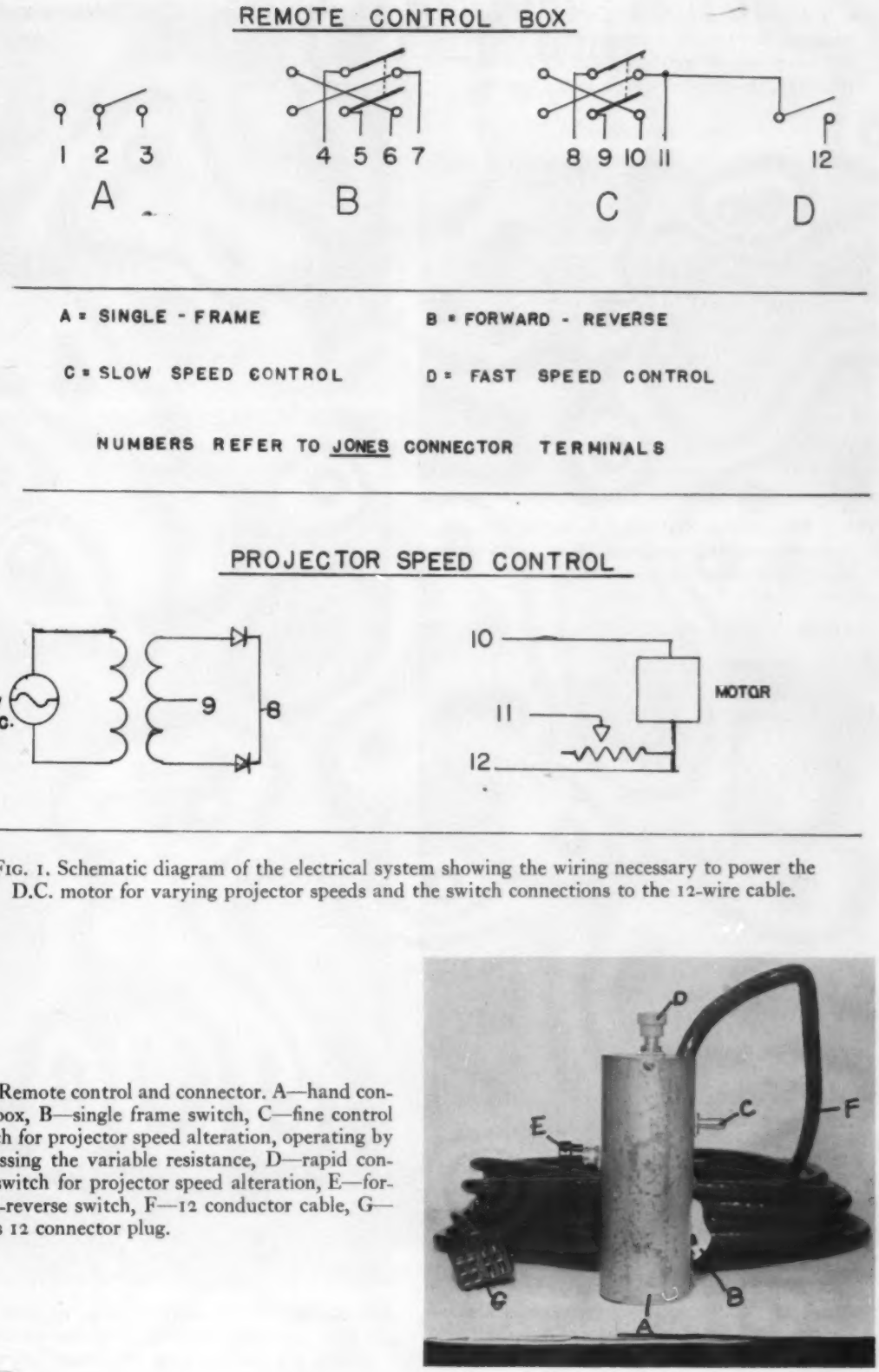
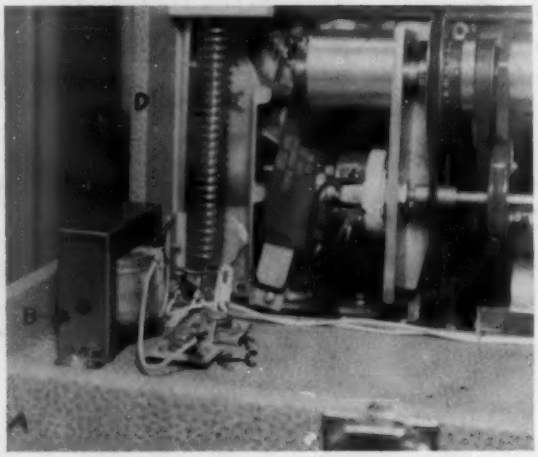


Fig. 1. Schematic diagram of the electrical system showing the wiring necessary to power the D.C. motor for varying projector speeds and the switch connections to the 12-wire cable.

Fig. 2. Remote control and connector. A—hand control box, B—single frame switch, C—fine control switch for projector speed alteration, operating by bypassing the variable resistance, D—rapid control switch for projector speed alteration, E—forward-reverse switch, F—12 conductor cable, G—Jones 12 connector plug.

Fig. 4. Power supply. A—hinged cover of projector housing, B—110-12 volt stepdown transformer, C—two selenium rectifiers wired for full wave rectification, D—front end of projector housing.



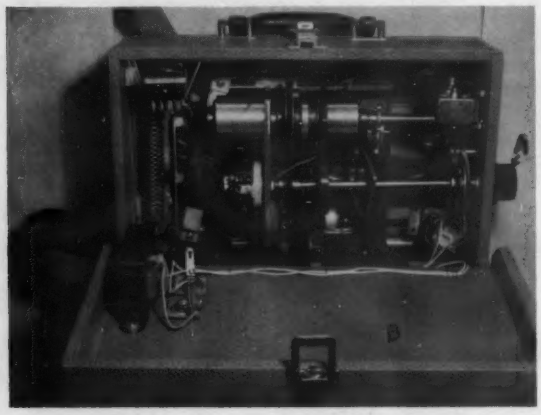


Fig. 3. Projector mechanism showing the location of component parts. A—manual speed control at rear of projector as available commercially, B—hinged cover of projector housing.

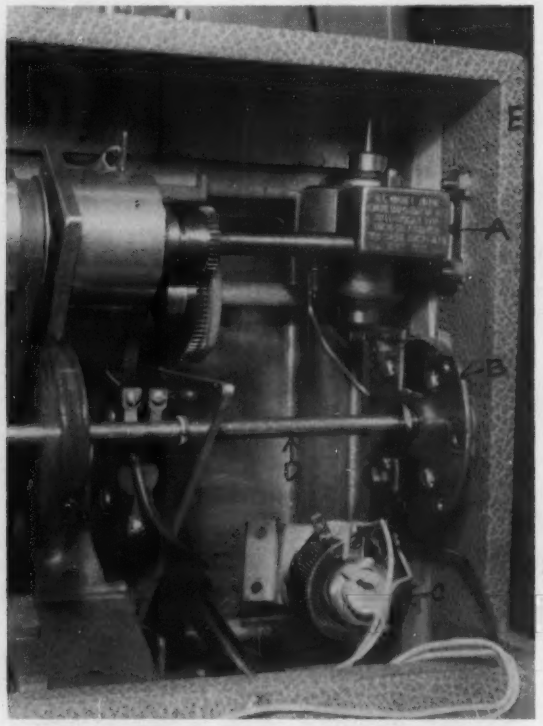


Fig. 5. Motor, gears and rheostat. A—D.C. permanent magnet motor, B—worm gear train, C—variable resistance to adjust speed of D.C. motor, D—projector speed rheostat control shaft, E—rear of projector housing.

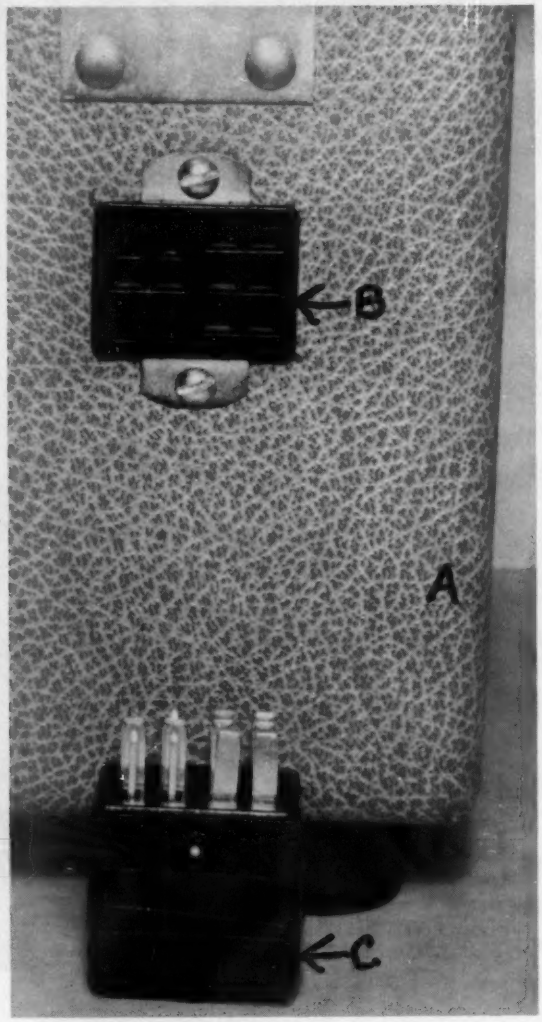


Fig. 6. Socket and shorting plug. A—rear of left side of projector housing, B—Jones 12 connector socket, C—shorting plug for manual operation.

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A PROTECTIVE LEAD APRON FOR THE CAUTIOUS RADIOLOGIST

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SOME years ago, after repeated measurements of radiation exposure showed that the shoulders and upper limbs during diagnostic procedures received high doses, we decided to design a protective apron that would give us adequate guarding. The excellent work of Archer[1] stimulated plans for an apron that was comparable to his, but less costly and of lighter weight.

The present model (Fig. 1) is the last of four which have been used routinely in our practice for the past five years. The protection it affords is as near perfect as we desire, the radiation reaching the inner side of the apron being of so low an order that it often approaches the limit of detection by the calibrated film and meters used.

Reference to Figure 1 will reveal the alarmingly high dose rates to the unprotected upper thorax and upper limbs. Our apron was designed mainly for the protection of these areas.

The weight of the apron is 20 pounds, too heavy for complete comfort but quite practical for the fluoroscopist in his routine work. The chief weight is supported by a

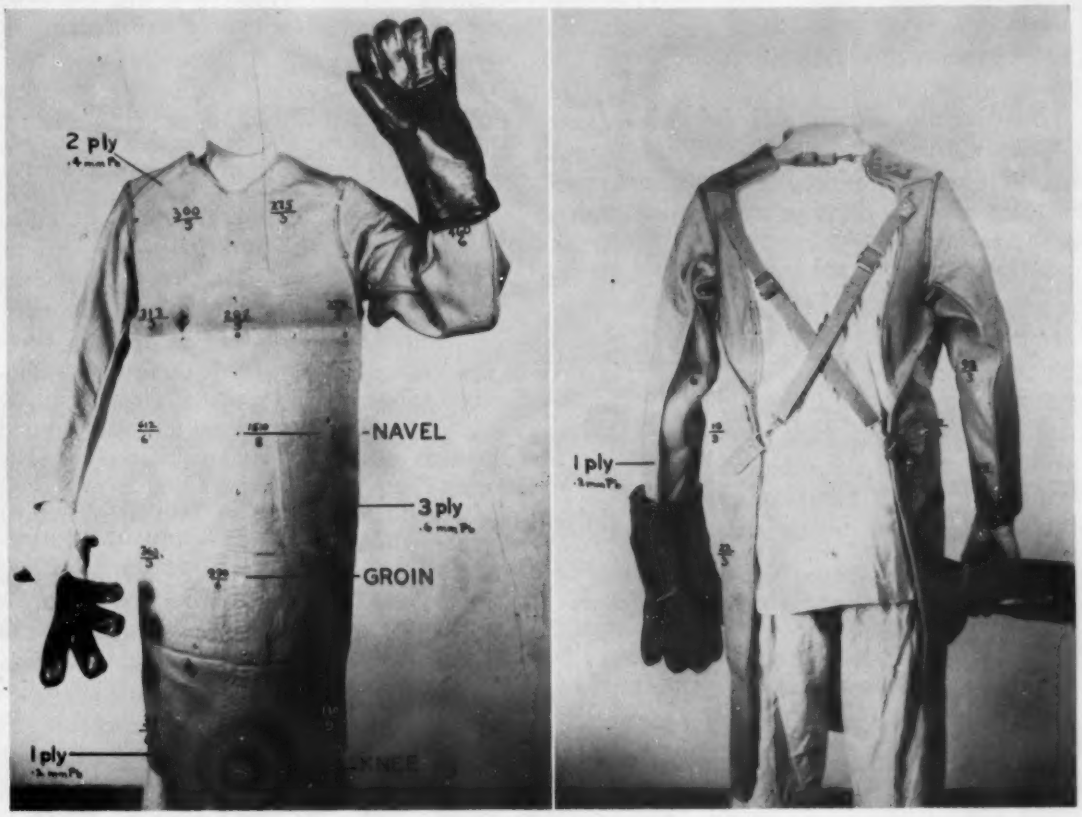

FIG. 1. Numerals on protective apron show mr of total exposure in 10 active days of fluoroscopy (326 min.).

$$\frac{\text{Numerator}}{\text{Denominator}} = \frac{\text{Outside Dose}}{\text{Inside Dose}}$$

short, snug transverse strap at the level of D-1.

The weight can be reduced about 5 pounds, in all probability, by using 1 ply on the thorax and at the lateral borders of the 3 ply mid-segment. This was not done in this model because of objection by the manufacturer.

However, with increased usage of kilovoltages over 100, the present apron is one calculated to provide, in addition to perspiration, peace of mind commensurate with its weight.

CONDITIONS OF TEST

Time and Current

19 days elapsed time	
10 active days.....	326.0 minutes of actual fluoroscopy
daily average.....	32.6 minutes of actual fluoroscopy
81 cases, average per case	4.0 minutes of actual fluoroscopy

Estimate of current: 326×2.5=815 ma. minutes

Roentgen-Ray Generating Equipment

a. Full wave rectified, 4 valve, 500 ma. generator, rotating anode tube under table: 85–90 kv., 2–3 ma., Al filter
   Output: 4.3 r per minute (table top—10×10 cm. aperture)
b. Full wave rectified, 4 valve, 200 ma. generator, rotating anode tube under table: 80–85 kv., 3 ma., 2 Al filter
   Output: 5.9 r per minute (table top—10×10 cm. aperture)

Protection to Radiologist

None but apron	
No side lead board No lead flaps on screen No table slot closure	Purposeful—to test maximum exposure

Labels on Apron

Label position same as test film: 2 sets of 21 each used to sample outside and inside of apron

$$\frac{\text{Numerator}}{\text{Denominator}} = \frac{\text{Outside Dose}}{\text{Inside Dose}}$$

Dose figures = total recorded in 10 active (19 elapsed) days for 326 minutes of actual fluoroscopy.

Calibrated Film Used

DuPont Dosimeter Type #558: 2 films in each packet
Fast #508 is of value from 0 to 700 mr
Slow #1290 is of value from 200 mr to 3 r

All films were developed and fixed simultaneously with controls included. The density was read in 3 areas of each film on a densitometer. A final record of the average of 3 readings was made. The variation of the 3 readings was a maximum of + or −.03 in a range of 0.1 to 0.48 for slow film and 0.5 to 2.8 for fast film. Graphs were made for each type of film and the most accurate portion of each curve was used.

Ionization Rate Meter Check

Exposure rates were spot checked by an ionization rate meter (Cutie Pie). The outside measurements ranged from 1.5 to 100.0 mr per hour. The inside measurements were 0.0 in all locations, except 1.0 mr per hour at #19 and #20.

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RADIATION DOSE TO THE SKIN IN ROENTGEN DIAGNOSTIC PROCEDURES*

OPTIMUM KVP. AND TISSUE MEASUREMENT TECHNIQUES

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THE increasing interest in long range biologic effects of radiation requires that more information with respect to radiation dosage be made available to workers in statistical research. Considerable investigation has been done in the past on the radiation dose being received from various diagnostic procedures. Handloser and Love,[4] Moore *et al.*,[6] Ritter *et al.*,[7] Ardran and Crooks,[1] Stanford and Vance,[8,9] Laughlin *et al.*,[5] and Billings *et al.*[2] are a few who have published papers on this subject.

Most of the reports in the literature contain data relating to various diagnostic techniques used in a particular department of radiology. For instance, the reports contain references to a certain peak kilovoltage, milliampere-seconds exposures, and filters that are employed for a given procedure. Frequently, the reports emanate from radiology departments where the techniques encompass good filtration of the roentgen rays and ample protection.

The work presented in this paper is aimed toward determining the skin radiation dose from diagnostic procedures with correct and incorrect filtration. Two methods were used: (1) the optimum kilovoltage technique as developed by Fuchs,[3] and (2) the tissue measurement technique, whereby the thickness of the part to be examined determines the kilovoltage to be used. The latter method is one in which the technician measures the patient, multiplies the thickness of the patient by 2, and adds 30 to obtain the kilovoltage to be employed for the procedure. Data obtained with both methods are presented.

MEASUREMENT TECHNIQUES

The radiation dose was determined with the use of a pressdwood phantom, a Model 570 Victoreen condenser r meter, a Model 326 10 r chamber and a Model 70 5 r and 25 r chamber for the 30 and 36 inch focal film distance (FFD) techniques. The 60 inch and the 72 inch FFD measurements were made with DuPont type 552 Dosimetry Film packets which had been calibrated for the peak kilovoltage and filtration used. The 10 r chamber and the 25 r chamber were of the medium energy range having a nominal wall thickness of 168 mg./cm.^{2} and 67 mg./cm.^{2} respectively. It was recognized that for the inherent filtration techniques, where very low half value layers exist, the medium energy range chambers did not give a measure of the total dose being delivered. Therefore, these chambers were compared with a Model 651 thin wall end window chamber having a nominal wall thickness of 7 mg./cm.^{2}. A correction curve was obtained for adjusting the results of the medium energy chambers to the true values that applied for the low half value layer radiation beams. The data contained in this report have corrections for the soft roentgen-ray beams where applicable.

Care was exercised in the use of the various ionization chambers not to exceed the intensity limits of the chamber. The 10 r chamber, when used with a Model 570 Victoreen r meter, has an upper intensity limit of 5 r per second before recombination becomes significant. This chamber was never used above an intensity of 4 r per

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second so that the measurements were well within the defined limits. The 25 r chamber, when used with a Model 570 Victoreen r meter, has an upper intensity limit of 45 r per second. This limit was never approached in any of the dose determinations made in this study.

For each diagnostic procedure, the radiation dose was determined with (1) inherent filtration only, (2) 1 mm. Al, (3) 2 mm. Al, and (4) 3 mm. Al added filters. The resultant roentgenogram in each instance is typical of those made in departments using the filter indicated.

RESULTS

The radiation dose delivered for each diagnostic procedure is shown in Tables I and II. Table I gives the radiation dose for the optimum kilovoltage technique as developed by Fuchs. Table II gives the radiation dose for the tissue measurement technique. The optimum kilovoltage technique may also be referred to as the long scale contrast technique and the tissue measurement technique as the short scale contrast technique. The difference between the two is that, in general, the Fuchs technique gives a greater latitude in the roentgenogram for deviations in procedure. The tissue measurement technique has a shorter latitude in the resultant roentgenogram.

In both tables, the radiation doses received during each diagnostic procedure, by a small, medium or large size patient, are given for exposures with inherent filtration only, and 1 mm. Al, 2 mm. Al, and 3 mm. Al added filtration. These data represent average values from three different machines in the Radiology Department at Duke University Medical Center. Two of the machines were Picker units and one was a Westinghouse unit.

DISCUSSION

The results are similar to those of other investigators. The proper use of filters markedly reduces the radiation dose being delivered for any diagnostic procedure. The amount of reduction that occurs in going from no filter to 3 mm. Al varies with the peak kilovoltage being used. However, in most instances the dose is reduced by a factor of 3. This factor alone should make it obvious to the user of roentgen-ray equipment that there is valid justification for the recommendation to use a total filtration of at least 2.5 mm. Al and preferably 3.5 mm. Al. (Assuming an inherent filtration of 0.5 mm. Al equivalent, this would be an added filter of 2.0 or 3.0 mm. Al respectively.) In many instances, not only does the filter reduce the roentgen-ray exposure but, due to the reduction of the scatter component, the resultant roentgenogram is better from a diagnostic standpoint.

One striking fact that is evident in such a comparison is that in the majority of procedures there is a greater dose delivered with the tissue measurement technique (short scale contrast) than with the optimum kilovoltage technique (long scale contrast). In many instances, the tissue measurement technique gives radiation doses as much as 3 times those of the optimum kilovoltage technique.

In view of these differences, one would question why diagnostic roentgenology has not shifted over completely to the optimum kilovoltage technique. In most instances, it is better to utilize the optimum kilovoltage technique for obtaining the best technical results. However, in our experience, the tissue measurement technique is indicated for soft tissue detail. A typical example of this is sinus roentgenography, in which soft tissue detail is important; if the optimum kilovoltage technique is used, soft tissue detail may be sacrificed. There must be a proper selection between these two methods. The selection is dependent upon whether soft tissue detail or bone detail is required. When a general diagnostic roentgenogram is indicated, in which it is necessary to visualize both bone detail and some soft tissue detail, a compromise must be made by sacrificing some of the soft tissue detail in order to obtain the bone detail. One point that is obvious from analyzing the data is that the added filtration of 2 to 3

TABLE I

LONG SCALE CONTRAST TECHNIQUE

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
Chest	Posteroanterior	18–20	3.33 S	80	14 × 17	72	.017	.009	.006	.005
		21–24	5 S	80	14 × 17	72	.025	.013	.009	.007
		25–27	6.66 S	80	14 × 17	72	.037	.019	.013	.010
	Oblique	19–23	5 S	80	14 × 17	72	.017	.013	.009	.007
		24–30	10 S	80	14 × 17	72	.056	.028	.019	.014
		31–35	15 S	80	14 × 17	72	.107	.054	.037	.027
	Lateral	–26	10 S	90	14 × 17	72	.064	.031	.021	.015
		27–32	13 S	90	14 × 17	72	.094	.044	.029	.021
		33–	15 S	90	14 × 17	72	.119	.052	.037	.026
Chest bronchi (w/opaque media)	Posteroanterior	20–24	10 SG	100	14 × 17	72	.074	.041	.026	.021
		25–27	15 SG	100	14 × 17	72	.115	.062	.040	.029
		28–30	20 SG	100	14 × 17	72	.159	.080	.055	.041
	Oblique	19–23	6.5 SG	100	14 × 17	72	.047	.025	.016	.014
		24–30	20 SG	100	14 × 17	72	.157	.082	.055	.040
		31–35	30 SG	100	14 × 17	72	.292	.130	.092	.067
	Lateral	24–27	15 SG	100	14 × 17	72	.115	.062	.041	.029
		28–29	25 SG	100	14 × 17	72	.199	.101	.069	.051
		30–34	30 SG	100	14 × 17	72	.291	.132	.092	.067
Chest bronchi (w/opaque media)	Posteroanterior	Child 2 to 10 yr.	10 SG	76	11 × 14	60	.0670	.0327	.0222	.0164
	Oblique		10 SG	82	11 × 14	60	.0697	.0354	.0244	.0183
	Lateral		10 SG	90	11 × 14	60	.0830	.0430	.0300	.0230
Chest bronchi (w/opaque media)	Posteroanterior	Infant	10 SG	70	8 × 10	60	.0562	.0265	.0155	.0128
	Oblique		10 SG	78	8 × 10	60	.0645	.0320	.0218	.0162
	Lateral		10 SG	86	8 × 10	60	.0727	.0374	.0259	.0197
Skull	Lateral	12	10 SG	85	10 × 12	36	.385	.177	.13	.093
		13–17	15 SG	85	10 × 12	36	.563	.346	.214	.158
		18	20 SG	85	10 × 12	36	.924	.458	.310	.218
	Posteroanterior	17	15 SG	85	8 × 10	36	.676	.330	.227	.162
		18–21	25 SG	85	8 × 10	36	1.21	.605	.398	.292
		22	35 SG	85	8 × 10	36	1.85	.912	.580	.442
	Anteroposterior	15	20 SG	85	8 × 10	36	.858	.406	.284	.210
		16–23	30 SG	85	8 × 10	36	1.44	.725	.476	.350
		24	40 SG	85	8 × 10	36	2.16	1.10	.698	.598
	Towne	17	40 SG	85	8 × 10	36	1.80	.885	.605	.436
		18–24	50 SG	85	8 × 10	36	2.56	1.26	.812	.642
		25 Cylinder cone	60 SG	85	8 × 10	36	3.39	1.40	1.06	.818
	Mento-Vertex	19	40 SG	85	8 × 10	36	1.90	.951	.634	.456
		20–25	50 SG	85	8 × 10	36	2.66	1.31	.835	.642
		26	60 SG	85	8 × 10	36	3.43	1.76	1.08	.810
Mastoid	Law	12	10 SG	85	8 × 10	30	.539	.240	.148	.115
		13–17	15 SG	85	8 × 10	30	1.02	.593	.285	.203
		18 Cylinder cone	20 SG	85	8 × 10	30	1.58	.665	.440	.320
	Stenvers	17	15 SG	85	8 × 10	30	1.017	.593	.285	.203
		18–21 Cylinder cone	20 SG	85	8 × 10	30	1.58	.665	.440	.320

F = Regular film with cardboard holder.
S = Medium speed screens, regular film.
G = 8:1 Lysholm grid and/or 8:1 Bucky grid.
SG = Medium speed screens and 8:1 Lysholm grid and/or 8:1 Bucky grid.
Technical Factors: Development, 5 min. at 68° F.; 2 mm. Al filtration; 4 valve tube full wave machine; cone to film.

TABLE I—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
Optic	Posteroanterior	17	10 SG	85	8 × 10	30	.676	.395	.190	.135
		18–21	20 SG	85	8 × 10	30	1.58	.665	.440	.320
		22 Cylinder cone	25 SG	85	8 × 10	30	2.27	.925	.600	.432
Mandible	Poasteranterior	17	10 SG	85	8 × 10	30	.676	.395	.190	.135
		18–21	15 SG	85	8 × 10	30	1.20	.497	.330	.240
		22 Cylinder cone	20 SG	85	8 × 10	30	1.81	.740	.500	.360
	Lateral	9	2.5 S	70	8 × 10	30	.094	.036	.023	.014
		10–14 Cylinder cone	5 S	70	8 × 10	30	.212	.085	.052	.035
Sinuses	Lateral	12	2.5 S	70	8 × 10	30	.097	.039	.024	.016
		13–17	5 S	70	8 × 10	30	.242	.092	.059	.039
		18 Cylinder cone	7.5 S	70	8 × 10	30	.451	.170	.105	.075
	Caldwell	17	20 S	70	8 × 10	30	.968	.368	.236	.156
		18–21	25 S	70	8 × 10	30	1.49	.565	.350	.250
		22 Cylinder cone	30 S	70	8 × 10	30	2.14	.780	.498	.360
	Sphenoid	17	20 S	70	8 × 10	30	.968	.368	.236	.156
		18–22	30 S	70	8 × 10	30	1.80	.678	.420	.300
		23 Cylinder cone	40 S	70	8 × 10	30	2.86	1.04	.665	.480
	Maxillary	17	20 S	70	8 × 10	30	.968	.368	.236	.156
		18–22	30 S	70	8 × 10	30	1.80	.678	.420	.300
		23 Cylinder cone	40 S	70	8 × 10	30	2.86	1.04	.665	.480
Cervical spine	Anteroposterior	11	10 SG	80	8 × 10	36	.325	.148	.102	.077
		12–15	15 SG	80	8 × 10	36	.566	.261	.171	.128
		16	20 SG	80	8 × 10	36	.805	.388	.254	.176
	Lateral	9	6.5 S	85	8 × 10	72	.040	.016	.012	.087
		10–13	15 S	85	8 × 10	72	.084	.038	.027	.019
		14	20 S	85	8 × 10	72	.108	.051	.034	.026
	Oblique	9	7.5 S	85	8 × 10	72	.047	.018	.014	.010
		10–13	15 S	85	8 × 10	72	.084	.038	.027	.019
		14	20 S	85	8 × 10	72	.108	.051	.034	.026
Dorsal spine	Anteroposterior	19	40 SG	75	7 × 17	36	1.56	.685	.462	.312
		20–24	60 SG	75	7 × 17	36	2.55	1.163	.733	.504
		25	80 SG	75	7 × 17	36	3.78	1.710	1.040	.768
	Lateral	30	50 SG	80	7 × 17	36	3.76	1.55	1.07	.750
		31–34	100 SG	80	7 × 17	36	8.25	3.37	2.32	1.60
		35	150 SG	80	7 × 17	36	14.3	6.0	3.75	2.62
Lumbar spine	Anteroposterior	17	60 SG	65	10 × 12	36	1.74	.625	.456	.294
		18–22	80 SG	70	10 × 12	36	2.90	1.23	.784	.520
		23	100 SG	70	10 × 12	36	4.02	1.74	1.050	.730
	Oblique	17	60 SG	75	8 × 10	36	2.15	.940	.638	.438
		18–22	80 SG	75	8 × 10	36	3.22	1.58	.940	.640
		23	100 SG	75	8 × 10	36	4.51	2.12	1.27	.90
	Lateral	26	100 SG	85	10 × 12	36	6.60	3.12	2.10	1.50
		27–32	200 SG	85	10 × 12	36	17.6	7.00	4.88	3.50
		33	250 SG	85	10 × 12	36	25.19	10.3	6.85	4.80

TABLE I—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
	Lateral	26	125 SG	85	8 × 10	36	7.15	3.67	2.25	1.79
	L-5	27–32	225 SG	85	8 × 10	36	18.59	7.88	5.50	3.94
		33	275 SG	85	8 × 10	36	27.72	10.3	7.80	5.28
		Cylinder cone								
Pelvis	Anteroposterior	15–19	80 SG	70	14 × 17	36	2.76	1.17	.768	.504
		20–23	100 SG	70	14 × 17	36	3.80	1.66	.99	.69
		24	125 SG	70	14 × 17	36	5.00	2.15	1.32	.913
Hip	Anteroposterior	15–19	80 SG	70	10 × 12	36	2.76	1.17	.768	.504
		20–23	100 SG	70	10 × 12	36	3.80	1.66	.99	.69
		24	125 SG	70	10 × 12	36	5.00	2.15	1.32	.913
	Lateral	19	125 SG	80	8 × 10	36	5.34	2.43	1.68	1.04
		20–24	150 SG	80	8 × 10	36	7.18	3.36	2.31	1.56
		25	175 SG	80	8 × 10	36	9.13	4.28	2.73	2.05
Abdomen	Anteroposterior	15–19	75 SG	70	14 × 17	36	2.58	1.10	.720	.473
		20–23	100 SG	70	14 × 17	36	3.80	1.66	.99	.69
		24–26	125 SG	70	14 × 17	36	5.00	2.15	1.32	.913
	Lateral	26	75 SG	80	14 × 17	36	3.87	1.83	1.17	.880
		27–32	100 SG	80	14 × 17	36	7.53	3.10	2.14	1.50
		33	125 SG	80	14 × 17	36	11.33	4.38	3.05	2.09
Gallbladder	Posteroanterior	14–17	20 SG	75	10 × 12	36	.720	.314	.213	.146
		18–22	30 SG	75	10 × 12	36	1.21	.594	.352	.240
		23–25	60 SG	75	10 × 12	36	2.70	1.27	.765	.540
Colon	Posteroanterior; anteroposterior	15–17	20 SG	85	14 × 17	36	.88	.420	.294	.210
		18–22	40 SG	85	14 × 17	36	1.98	.983	.642	.477
		23–25	80 SG	85	14 × 17	36	4.35	2.21	1.39	1.09
	Air contrast	15–17	5 SG	95	14 × 17	36	.280	.142	.097	.078
		18–21	10 SG	95	14 × 17	36	.620	.334	.212	.174
		22–25	20 SG	100	14 × 17	36	1.45	.860	.520	.415
	Chassard Lapine	Small	50 SG	100	14 × 17	36	3.75	2.26	1.28	1.13
		Medium	80 SG	100	14 × 17	36	8.58	4.16	2.80	2.16
		Large	100 SG	100	14 × 17	36	13.75	6.10	4.14	2.95
Pelvis (Pelvimetry)	Anteroposterior; supine	20–23	100 SG	75	14 × 17	36	4.46	1.94	1.22	0.84
		24–28	150 SG	75	14 × 17	36	7.09	3.21	1.95	1.43
		29–33	200 SG	75	14 × 17	36	13.64	5.24	3.68	2.52
Pelvis	Anteroposterior Thoms	Small	100 SG	100	10 × 12	36	7.21	3.94	2.45	2.05
		Medium	150 SG	100	10 × 12	36	11.28	6.79	3.83	3.39
		Large	200 SG	100	10 × 12	36	21.45	10.40	7.00	5.40
	Lateral	Small	80 SG	100	14 × 17	36	5.80	3.44	2.08	1.64
		Medium	100 SG	100	14 × 17	36	9.62	4.87	3.32	2.47
		Large	150 SG	100	14 × 17	36	18.15	8.26	5.80	4.20
Placenta	Lateral	Small	12.5 SG	100	14 × 17	36	.907	.538	.325	.256
		Medium	25 SG	100	14 × 17	36	2.41	1.22	.830	.620
		Large	40 SG	100	14 × 17	36	4.84	2.20	1.54	1.12
Hand	Posteroanterior	2	40 F	50	8 × 10	36	.588	.168	.092	.074
		3–5	50 F	50	8 × 10	36	.761	.225	.125	.100
		6	60 F	50	8 × 10	36	.957	.287	.162	.126
	Lateral	7–10	120 F	50	8 × 10	36	1.958	.61	.36	.264
		11	150 F	50	8 × 10	36	2.68	.88	.51	.36
Shoulder	Anteroposterior	9–11	7.5 SG	80	8 × 10	36	.249	.108	.075	.053
		12–16	12.5 SG	80	8 × 10	36	.478	.217	.146	.105
		17–18	15 SG	80	8 × 10	36	.640	.291	.201	.132

TABLE I—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
	Lateral	5	60 F	60	10 × 12	36	1.15	.36	.24	.18
		6–9	70 F	60	10 × 12	36	1.46	.455	.336	.228
		10	80 F	60	10 × 12	36	1.64	.687	.384	.256
Os calcis	Axial	4–8	60 F	80	8 × 10	36	1.95	.75	.55	.398
		9–13	100 F	80	8 × 10	36	3.52	1.56	1.13	.75
		14–18	125 F	80	8 × 10	36	5.02	2.30	1.60	1.10
Tibia-fibula	Anteroposterior	8	5 SG	80	7 × 17	36	.170	.065	.05	.034
		9–12	10 SG	80	7 × 17	36	.33	.144	.100	.070
		13	15 SG	80	7 × 17	36	.574	.261	.176	.126
	Lateral	7	2.5 SG	80	7 × 17	36	.077	.031	.022	.017
		8–11	3.2 SG	80	7 × 17	36	.105	.046	.032	.023
		12	5 SG	80	7 × 17	36	.574	.078	.056	.038
Knee	Anteroposterior	9	5 SG	80	8 × 10	36	.170	.065	.05	.034
		10–14	6.5 SG	80	8 × 10	36	.229	.101	.073	.049
		15	10 SG	80	8 × 10	36	.401	.184	.128	.088
	Lateral	8	5 SG	80	8 × 10	36	.170	.065	.05	.034
		9–13	6.5 SG	80	8 × 10	36	.229	.101	.073	.049
		14	7.5 SG	80	8 × 10	36	.301	.138	.096	.066
Stomach (with barium)	Right antero-posterior	15–17	20 SG	90	10 × 12	36	1.02	.500	.340	.262
		18–22	40 SG	85	10 × 12	36	2.02	.982	.641	.477
		23–26	80 SG	85	10 × 12	36	4.34	2.21	1.38	1.09

TABLE II

SHORT SCALE CONTRAST TECHNIQUE*

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
Chest	Posteroanterior	18	10 S	62	14 × 17	72	.037	.017	.011	.008
		21	10 S	68	14 × 17	72	.045	.021	.014	.010
		27	10 S	78	14 × 17	72	.054	.027	.018	.014
	Oblique	19	20 S	64	14 × 17	72	.079	.037	.024	.011
		24	20 S	74	14 × 17	72	.097	.047	.032	.023
		31	20 S	90	14 × 17	72	.137	.071	.050	.038
	Lateral	26	20 S	78	14 × 17	72	.109	.054	.037	.027
		29	20 S	84	14 × 17	72	.122	.062	.043	.033
		32	20 S	90	14 × 17	72	.137	.071	.050	.038

F = Regular film with cardboard holder.
NS = Non screen film with cardboard holder.
S = Medium speed screens.
SG = Medium speed screens and 8:1 Lysholm and/or 8:1 Bucky grid.
Technical Factors: Development, 5 min. at 68° F.; 2 mm. Al filtration; rotating anode tube; 4 valve tube full wave machine; cone to film.
* The kilovoltage variation per centimeter thickness was determined by the formula kv. peak = 2 × cm. thickness + 30.

TABLE II—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
Chest bronchi (w/opaque media)	Posteroanterior	18	10 S	67	14 × 17	72	.043	.020	.013	.010
		21	10 S	73	14 × 17	72	.046	.022	.015	.011
		27	10 S	83	14 × 17	72	.058	.030	.020	.015
	Oblique	19	20 S	70	14 × 17	72	.090	.042	.028	.021
		24	20 S	82	14 × 17	72	.116	.059	.040	.031
		31	20 S	95	14 × 17	72	.144	.076	.052	.040
	Lateral	26	20 S	85	14 × 17	72	.119	.061	.042	.032
		27	20 S	90	14 × 17	72	.130	.068	.048	.036
		32	20 S	98	14 × 17	72	.152	.080	.057	.043
Skull	Lateral	12	60 SG	56	10 × 12	36	1.17	.432	.258	.144
		14	60 SG	60	10 × 12	36	1.36	.540	.318	.228
		18	60 SG	68	10 × 12	36	1.98	.842	.546	.348
	Posteroanterior	17	100 SG	66	8 × 10	36	3.00	1.26	.81	.53
		19	100 SG	70	8 × 10	36	3.53	1.53	.98	.64
		22	100 SG	76	8 × 10	36	4.32	2.02	1.28	.88
	Anteroposterior	15	100 SG	62	8 × 10	36	2.53	1.00	.62	.43
		19	100 SG	70	8 × 10	36	3.53	1.53	.98	.64
		24	100 SG	80	8 × 10	36	4.96	2.32	1.50	1.10
	Towne	17	125 SG	66	8 × 10	36	3.75	1.57	1.01	.662
		19	125 SG	70	8 × 10	36	4.42	1.91	1.10	.80
		25	125 SG	82	8 × 10	36	6.55	3.34	1.74	1.61
	Mento-Vertex	19	125 SG	70	8 × 10	36	4.43	1.91	1.10	.80
		21	125 SG	74	8 × 10	36	5.09	2.30	1.45	.988
		26	125 SG	84	8 × 10	36	8.35	3.49	2.15	1.77
Mastoid	Law	12	60 SG	56	8 × 10	30	1.75	.60	.336	.210
		14	60 SG	60	8 × 10	30	2.18	.770	.462	.282
		17 Cylinder cone	60 SG	66	8 × 10	30	2.88	1.056	.661	.432
	Stenvers	17	80 SG	66	8 × 10	30	3.85	1.408	.881	.576
		18	80 SG	68	8 × 10	30	4.20	1.58	.994	.680
		21 Cylinder cone	80 SG	74	8 × 10	30	5.49	2.18	1.375	.978
Optic	Posteroanterior	17	80 SG	66	8 × 10	30	3.85	1.408	.881	.576
		18	80 SG	68	8 × 10	30	4.20	1.58	.994	.680
		21	80 SG	74	8 × 10	30	5.49	2.18	1.375	.978
Mandible	Posteroanterior	15	100 SG	62	8 × 10	30	3.97	1.40	.86	.53
		18	100 SG	88	8 × 10	30	5.25	1.98	1.24	.85
		21	100 SG	74	8 × 10	30	6.85	2.73	1.72	1.22
	Lateral	9	10 S	50	8 × 10	30	.226	.067	.036	.022
		10	10 S	52	8 × 10	30	.245	.074	.072	.026
		12 Cylinder cone	10 S	56	8 × 10	30	.294	.100	.096	.035
Sinuses	Lateral	9	20 S	36	8 × 10	30	.303	.074	.034	.012
		13	20 S	44	8 × 10	30	.520	.142	.072	.044
		16 Cylinder cone	20 S	50	8 × 10	30	.560	.168	.096	.050
	Caldwell	17	80 S	66	8 × 10	30	3.85	1.408	.881	.576
		18	80 S	68	8 × 10	30	4.20	1.58	.994	.680
		22 Cylinder cone	80 S	76	8 × 10	30	6.02	2.30	1.52	1.04

TABLE II—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
	Maxillary	17	80 S	66	8 × 10	30	3.85	1.408	.881	.576
		18	80 S	68	8 × 10	30	4.20	1.58	.994	.680
		22	80 S	76	8 × 10	30	6.02	2.30	1.52	1.04
Cervical spine	Anteroposterior	10	100 SG	52	8 × 10	36	1.83	.59	.32	.23
		13	100 SG	58	8 × 10	36	2.15	.81	.48	.34
		16	100 SG	64	8 × 10	36	2.68	1.12	.70	.48
	Lateral	9	40 S	50	8 × 10	72	.096	.041	.023	.015
		12	40 S	56	8 × 10	72	.121	.053	.032	.022
		14	40 S	60	8 × 10	72	.136	.062	.039	.027
	Oblique	9	40 S	50	8 × 10	72	.096	.041	.023	.015
		12	40 S	56	8 × 10	72	.121	.053	.032	.022
		14	40 S	60	8 × 10	72	.136	.062	.039	.027
Dorsal spine	Anteroposterior	19	100 SG	70	7 × 17	36	3.53	1.53	.98	.64
		21	100 SG	74	7 × 17	36	4.07	1.84	1.16	.79
		25	100 SG	82	7 × 17	36	5.23	2.67	1.65	1.29
	Lateral	30	150 SG	66	7 × 17	36	4.50	1.89	1.21	.797
		32	150 SG	70	7 × 17	36	5.30	2.30	1.47	.960
		35	150 SG	76	7 × 17	36	6.48	3.03	1.92	1.32
Lumbar spine	Anteroposterior	17	100 SG	66	10 × 12	36	3.00	1.26	.81	.53
		19	100 SG	70	10 × 12	36	3.53	1.53	.98	.64
		23	100 SG	78	10 × 12	36	4.72	2.17	1.39	.99
	Oblique	17	100 SG	66	10 × 12	36	3.00	1.26	.81	.53
		19	100 SG	70	10 × 12	36	3.53	1.53	.98	.64
		23	100 SG	78	10 × 12	36	4.72	2.17	1.39	.99
	Lateral	26	200 SG	84	10 × 12	36	11.12	5.84	3.60	2.96
		28	200 SG	88	10 × 12	36	15.80	7.50	5.10	3.50
		33	200 SG	98	10 × 12	36	25.3	11.50	9.90	5.72
	Spot L-5	26	250 SG	84	8 × 10	33	21.2	9.05	6.20	4.33
		28	250 SG	88	8 × 10	33	26.0	10.4	7.22	5.73
		33	250 SG	98	8 × 10	33	38.7	18.20	12.7	9.85
Pelvis	Anteroposterior	15	100 SG	62	14 × 17	36	2.49	1.0	.62	.43
		20	100 SG	72	14 × 17	36	3.74	1.66	1.05	.70
		24	100 SG	80	14 × 17	36	4.98	2.32	1.50	1.10
Hip	Anteroposterior	15	100 SG	62	14 × 17	36	2.49	1.0	.62	.43
		20	100 SG	72	14 × 17	36	3.74	1.66	1.05	.70
		24	100 SG	80	14 × 17	36	4.98	2.32	1.50	1.10
	Lateral	19	100 SG	70	8 × 10	36	3.53	1.53	.98	.64
		22	100 SG	76	8 × 10	36	4.30	2.02	1.28	.88
		25	100 SG	82	8 × 10	36	5.22	2.67	1.65	1.29
Abdomen	Anteroposterior	15	80 SG	62	14 × 17	36	1.99	.80	.496	.344
		20	80 SG	72	14 × 17	36	3.99	1.33	.842	.560
		25	80 SG	82	14 × 17	36	4.18	2.13	1.32	1.03
	Lateral	26	80 SG	78	14 × 17	36	3.78	1.74	1.10	.792
		28	80 SG	82	14 × 17	36	5.65	2.60	1.68	1.16
		32	80 SG	92	14 × 17	36	8.35	3.70	2.56	1.84
Gallbladder	Posteroanterior	14	80 SG	60	10 × 12	36	1.82	.720	.424	.304
		19	80 SG	70	10 × 12	36	2.83	1.23	.784	.512
		23	80 SG	78	10 × 12	36	3.78	1.74	1.11	.792

TABLE II—(*Continued*)

Region	Projection	Thickness Range (cm.)	Mas.	Kv. Peak	Film Size	FFD (in.)	Skin Dose in (r)			
							No Filter (corrected)	1 mm. Al	2 mm. Al	3 mm. Al
Colon (air contrast)	Anteroposterior; posteroanterior	16	20 SG	88	14 × 17	36	.979	.468	.320	.240
		21	40 SG	88	14 × 17	36	2.13	1.08	.70	.536
		27	80 SG	90	14 × 17	36	5.75	2.84	1.92	1.38
Pelvis (Pelvimetry)	Anteroposterior; supine	20	100 SG	72	14 × 17	36	3.74	1.66	1.05	.70
		28	100 SG	88	14 × 17	36	7.76	3.75	2.54	1.75
		33	100 SG	98	14 × 17	36	13.1	5.62	3.90	2.85
Pelvis	Anteroposterior	24	200 SG	80	10 × 12	36	9.94	4.64	3.00	2.20
		30	200 SG	92	10 × 12	36	18.7	8.60	5.80	4.30
	Thoms	35	200 SG	100	10 × 12	36	27.5	12.2	8.28	5.80
	Lateral	26	200 SG	84	10 × 12	36	11.1	5.84	3.60	2.96
		30	200 SG	92	10 × 12	36	18.7	8.60	5.80	4.30
		34	200 SG	100	10 × 12	36	27.5	12.2	8.28	5.80
Hand	Posteroanterior	2	50 NS	36	8 × 10	36	.403	.10	.04	.02
		4	50 NS	40	8 × 10	36	.515	.14	.06	.03
		6	50 NS	44	8 × 10	36	.625	.18	.10	.06
	Lateral	7	50 NS	46	8 × 10	36	.678	.20	.10	.06
		11	50 NS	52	8 × 10	36	.915	.29	.16	.12
Shoulder	Anteroposterior	9	50 NS	52	8 × 10	36	.915	.29	.16	.12
		12	50 NS	56	8 × 10	36	.975	.36	.215	.145
		17	50 NS	66	8 × 10	36	1.50	.63	.405	.265
Os calcis	Axial	7	50 NS	46	8 × 10	36	.678	.20	.10	.06
		10	50 NS	52	8 × 10	36	.915	.29	.16	.12
		15	50 NS	62	8 × 10	36	1.24	.50	.31	.215
Tibia-fibula	Anteroposterior	8	10 S	48	7 × 17	36	.150	.045	.024	.016
		11	10 S	52	7 × 17	36	.184	.059	.032	.023
		13	10 S	58	7 × 17	36	.216	.081	.048	.034
	Lateral	7	10 S	46	7 × 17	36	.136	.040	.021	.013
		10	10 S	52	7 × 17	36	.184	.059	.032	.023
		12	10 S	56	7 × 17	36	.196	.072	.043	.029
Knee	Anteroposterior	9	10 S	48	8 × 10	36	.150	.045	.024	.016
		11	10 S	56	8 × 10	36	.208	.073	.047	.031
		15	10 S	62	8 × 10	36	.248	.100	.062	.043
	Lateral	8	10 S	48	8 × 10	36	.150	.045	.024	.016
		10	10 S	52	8 × 10	36	.184	.059	.032	.023
		14	10 S	60	8 × 10	36	.228	.090	.053	.038
Stomach (w/barium)		18	40 SG	82	10 × 12	36	1.76	.862	.577	.392
		21	80 SG	88	10 × 12	36	4.27	2.16	1.40	1.07
		25	100 SG	86	10 × 12	36	6.75	3.25	2.18	1.55

mm. Al is advisable regardless of which of the two procedures is used.

One of the major purposes of conducting such a study as this is to have data available so that long range effects of irradiation may be determined. This means that radiation dose values should be available for any given diagnostic procedure used and a radiation logging system for patients should be adopted.

The problem, of course, is far more complex than that of skin dose alone. The integral dose is far more important, and the determination of this is quite difficult. For instance, in determining the integral dose or total energy absorbed, one must know the half value thickness in tissue for each technique being used, and the actual volume being irradiated. Consequently, a large amount of data should be accumulated. Most diagnostic routines are set up for a focal film distance and not a focal skin

distance technique. The half value thickness differs with varying thicknesses of patients, due to the variation in focal skin distance. The determination for each technique of the actual volume being irradiated would vary considerably between patients due to differences in focal skin distances and scattering conditions. This combination of variables makes the problem appear insurmountable as far as establishing any constants is concerned. However, one cannot help but feel that necessary measurements can and should be made. Such measurements should be based upon estimates obtained under test conditions that closely approximate those actually being used.

SUMMARY

The radiation doses being received during various routine roentgen diagnostic procedures have been determined. These doses were determined for filtration conditions ranging from good to poor using the optimum kilovoltage technique and the tissue measurement technique. A short discussion of the results is given.

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* *Ex officio.*

EDITORIALS

INSTRUCTION COURSES OF THE SIXTY-FIRST ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

PRESIDENT-ELECT Harold Reineke has requested that we continue the Instruction Courses for the 1960 Meeting in Atlantic City. This will mark the twenty-first year since these courses were started during the term of President Dr. Edward Jenkinson and under the direction of the late Dr. B. R. Kirklin. The modus operandi established by Dr. Kirklin and modified slightly by his successor Dr. Harry Weber has functioned so well that we have not felt any need to alter it. Consequently, the program this year will follow the same general pattern of the past twenty years.

The schedule of the entire program including brief abstracts of each course appears in this issue of the JOURNAL. It is possible to select the courses of your choice from this published information and register in advance of the meeting. If your registration reaches the Director prior to September 17, the tickets will be mailed to you before the meeting. It is also possible to obtain tickets in Atlantic City beginning September 26 through September 30. Since the rooms in which the courses will be held are not large, it is probable that some courses may be completely filled before the meeting starts.

As has been customary since these courses were started, a fee of $2.00 per course will be charged to nonmembers of the American Roentgen Ray Society. This fee does not apply to those physicians or graduate students who are in training in Radiology or related fields.

Approximately two-thirds of the courses are devoted to diagnostic subjects and the rest deal with therapy, physics and nuclear medicine. We attempt to obtain instructors who are well qualified and experienced in their particular subject. We also change some of the faculty each year in order to vary the program and keep abreast of the newer developments.

This year we are particularly pleased to have on our faculty Dr. Robert McWhirter of Edinburgh, Scotland. Since he is internationally well known for his work on cancer of the breast, it was felt that many more physicians would want to hear him than can be accommodated in the regular instruction course rooms. Consequently, the hotel has made available a much larger room (the 22 Club) at 1:30 P.M. on Wednesday, September 28. No fee will be charged for this course.

We have been assured that all the instruction course rooms will be air conditioned. These rooms are all located on the conference floor of the Ambassador Hotel, one flight above the lobby level. The 22 Club is located on the ground floor which is one flight below the lobby level. Complete information concerning the courses and remaining tickets can be obtained at a desk which will be located near the main registration area.

It is our hope that the growing popularity and educational value of these courses will continue this year. Our sincere thanks are extended to the fifty-two instructors who give so freely of their knowledge and time in preparing and presenting their material.

HAROLD O. PETERSON, M.D.
Director, Section on Instruction

DONALD SMYTHE CHILDS
1888–1960

"To live in hearts we leave behind is not to die."
Thomas Campbell

THE death of Donald S. Childs on April 27, 1960, closes a memorable chapter in the annals of American radiology. A consideration of his many activities cannot fail to indicate his profound influence on those with whom he came in contact—his family, of whom he was justly proud, his professional colleagues, his business associates, the companions who shared his love of the out-of-doors, and a host of

persons in all walks of life who called him friend. Nor was this influence limited to individuals. It extended to the specialty to which he devoted his life and beyond that to medicine in general.

Dr. Childs was born in Syracuse, New York, January 5, 1888, the son of Henry Childs and Laura Smythe. He received his education in the public schools of Syracuse and in 1909 was graduated from the school of Medicine of Syracuse University, with which he was destined to be associated for most of his life. He became Professor of Radiology in the College of Medicine of Syracuse University in 1914 and maintained this position until 1948, when he was appointed Emeritus Professor. He taught osteology in the same school for twenty-eight years, holding the title, Professor of Osteology. Because of this experience he was exceptionally well versed in the radiological study of the skeletal system, especially the spine.

For forty years Dr. Childs was radiologist to St. Mary's Maternity Hospital of Syracuse and for thirty years radiologist for the Elmcrest Children's Center and The House of Providence. In addition, he served several other hospitals in the capacity of a consultant. He was a Captain in the Medical Corps of the United States Army during World War I.

Dr. Childs became a member of the American Roentgen Ray Society in 1930. He was also a member of The Radiological Society of North America and is perhaps best known for his activities as Secretary-Treasurer of that organization, and as Business Manager of *Radiology*, its official journal, which positions he held from 1931 until his death. These duties he took over at a time of crisis in the history of both the Society and its journal, and by his judicious management and sound financial practices he brought them to the place they occupy today. In 1949 he was awarded the Gold Medal of the Radiological Society of North America for his outstanding cont.ibutions to the science of Radiology, and on the occasion of his 25th anniversary as Secretary-Treasurer of the Radiological Society he was presented with a silver tray inscribed with the names of all the past presidents during his regime.

He was also a fellow of the American College of Radiology, Chairman of the Section of Radiology of the American Medical Association in 1956–1957, a Trustee of the American Board of Radiology, from 1940 to 1958, and President of the Board in 1957–1958. He was a former President of the Academy of Medicine of Syracuse. He took a special interest in the Blue Cross and Blue Shield plans of Syracuse from the time of their inception and served as a member of the Board of Directors for each of these organizations until his death.

In his capacity as Secretary-Treasurer of the Radiological Society of North America, Dr. Childs was responsible in large part for the success of the annual meetings of that organization. Few outside of those immediately concerned with the arrangements were aware of the careful study and attention which he gave to the minutest details. Over the years he had perfected a technique of planning that was reflected in the growing popularity of these meetings.

Dr. Childs' knowledge of legal affairs was widely recognized, and his advice was frequently sought by attorneys and insurance company executives. His familiarity with medicolegal problems was also of great advantage in his dealing with the problems of organized medicine.

What has been said relates chiefly to Dr. Childs in his professional capacity. There are many facets which reflect the joy of living and richness of interests of Don Childs the man. From his college days, music and the theater gave him especial pleasure. As a baseball fan he usually made it a point to be in Florida during the spring training season. He was a close follower of the Syracuse University football team. Outdoor life in general and fishing in particular were interests that filled his occasional leisure. As much time as was available he spent with his family at their summer home on Fourth Lake in the Adiron-

dacks, pursuing these hobbies with his usual verve.

Dr. Childs is survived by his wife Amy Edwards, his two sons, Dr. Donald S. Childs, Jr., of Rochester, Minnesota, and Eleazer, or Lee, of Syracuse, and by nine grandchildren.

Don Childs was a man whose advice and help to others secured for him the friendship of persons both in high places and in the humbler stations of life. He was firm in situations which required firmness, but beneath this outer façade was a kind and friendly spirit constantly working for the good of American Radiology. It was my privilege to work in close harmony with him for twenty-five years and my affection and respect for him increased constantly over the period of our association.

Howard P. Doub, M.D.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*
Donn G. Mosser, M.D., *Associate Director*

Titles and Abstracts of Courses Offered

Sixty-First Annual Meeting
Ambassador Hotel, Atlantic City, New Jersey
September 27 to September 30, 1960

The Instruction Courses for the Sixty-first Annual Meeting of the American Roentgen Ray Society will all be of ninety minutes' duration and will be held between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M. on Friday. No other official activity has been scheduled for this time.

Fourteen rooms are available for the courses and one course will be offered in each room each afternoon of the Annual Meeting. A total of fifty-two instructors will give fifty-six courses during the four days.

There will be thirty-nine courses in Diagnostic Roentgenology and seventeen courses in Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology. Of the Diagnostic courses seventeen are single period courses and eleven are two period courses. In Therapy and Nuclear Medicine there is only one two period course. It is desirable but not essential to register for both periods of a two period course.

A special course will be given by Dr. R. McWhirter, Consultant, South-Eastern Regional Radiotherapy Service, Royal Infirmary and Western General Hospital, Edinburgh, Scotland, on Wednesday, September 28, at 1:30 P.M. at the 22 Club, which is located on the ground floor, one flight below the lobby level. *No tickets* will be required for this course.

GENERAL INFORMATION

Location of Courses

All courses with the exception of the special course of Dr. McWhirter will be held in rooms which are located on the conference floor, one flight above the lobby level. Full information will be available at the desk of the Section on Instruction, which will be located in the General Registration Area on Monday afternoon and from 8:30 A.M. until 4:30 P.M. Tuesday, Wednesday, Thursday and Friday.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only.

Following the list of titles and abstracts is a general order sheet. First, second and third choices for each period should be selected carefully and indicated on the order sheet. It is estimated that the number who can attend each course each day will range between 30 and 100 persons depending on room size. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized.

It is possible for one to attend only four periods of instruction, so the condensed schedule given on the last pages of this program should be consulted with care when registering and ordering tickets for the courses.

Reservations for the courses will be made in the order in which the pink order sheets are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $2.00 per period. Full-time graduate students in radiology will be admitted without fee, but they will be required to make application for the courses they wish to attend.

Prior to September 17, 1960, the order forms should be sent to the Director, Dr. Harold O. Peterson, 1995 West County Road "B," St. Paul 13, Minnesota. After September 17, 1960, the order forms should be sent to Dr. Harold O. Peterson, Ambassador Hotel, Atlantic City, New Jersey.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 17, 1960. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the Registration Desk of the Section on Instruction, Monday afternoon, September 26, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An attendant will be at the door of each conference room to collect the tickets.

THE FACULTY

1. Ernest Aegerter, M.D., Professor Pathology, Temple University School of Medicine, Philadelphia, Pennsylvania.
2. J. Robert Andrews, M.D., D.Sc. (Med.), Radiation Branch, National Cancer Institute, National Institutes of Health, Public Health Service, Department of Health, Education and Welfare, Bethesda, Maryland.
3. Arnold L. Bachman, M.D., Assistant Professor of Radiology, Columbia University, New York, New York.
4. Jean Bouchard, M.D., Department of Radiology, Royal Victoria Hospital, Montreal, Quebec, Canada.
5. John A. Campbell, M.D., Chairman, Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.
6. George Casarett, Ph.D., Department of Radiology, University of Rochester School of Medicine, Rochester, New York.
7. M. Y. Colby, Jr., M.D., Consultant, Department of Radiotherapy, Mayo Clinic, Rochester, Minnesota.
8. Juan A. del Regato, M.D., Director, Penrose Cancer Hospital, Colorado Springs, Colorado.
9. Gerald D. Dodd, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
10. Robert L. Egan, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
11. Lewis E. Etter, M.D., Director, Department of Radiology, The Falk Clinic; Consultant, Western Psychiatric Institute and Clinic; Professor of Radiology, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.
12. William R. Eyler, M.D., Chairman, Department of Radiology, Henry Ford Hospital, Detroit, Michigan.
13. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.
14. Nathaniel Finby, M.D., Department of Radiology, The New York Hospital-Cornell Medical Center, New York, New York.
15. Felix G. Fleischner, M.D., Director, Department of Radiology, Beth Israel Hospital; Clinical Professor of Radiology, Harvard University, Boston, Massachusetts.
16. Manuel Garcia, M.D., Director, Department of Therapeutic Radiology, Charity Hospital of Louisiana, New Orleans, Louisiana.
17. Brit B. Gay, Jr., M.D., Department of Radiology, Emory University, Atlanta, Georgia.
18. Ruth Guttmann, M.D., Director, Radiation Therapy, Francis Delafield Hospital, New York, New York.
19. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
20. George Jacobson, M.D., Professor and Head of Department of Radiology, University of Southern California, Los Angeles, California.
21. Harold G. Jacobson, M.D., Chief of the Division of Diagnostic Radiology at Montefiore Hospital; Clinical Professor of Radiology at New York University College of Medicine; Senior Con-

sultant in Radiology to the Veterans Hospital in the Bronx, New York, New York.

22. John Kirkpatrick, M.D., Department of Radiology, St. Christopher's Hospital, Philadelphia, Pennsylvania.

23. Maurice Lenz, M.D., Consultant in Radiotherapy, Presbyterian Hospital; Emeritus Professor of Radiology, Columbia University College of Physicians and Surgeons, New York, New York.

24. Richard G. Lester, M.D., Associate Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

25. Emanuel J. Levin, M.D., Director of Radiology, Maimonides Hospital of Brooklyn; Professor of Radiology, State University of New York, Brooklyn, New York.

26. Gisèle Lussier, M.D., Department of Radiology, Hotel Dieu Hospital, Montreal, Quebec, Canada.

27. J. C. F. MacDonald, M.A., Ph.D., Senior Physicist, The Ontario Cancer Foundation, London Clinic, Victoria Hospital, London, Ontario, Canada.

28. Alexander Stewart Macmillan, M.D., Director of Radiology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

29. Richard H. Marshak, M.D., Associate Radiologist, Mt. Sinai Hospital, New York, New York.

30. Charles L. Martin, M.D., The Martin X-ray and Radium Clinic, Dallas, Texas.

31. Professor R. McWhirter, M.B., Ch.B., F.R.C.S. (Edin.), F.F.R., D.M.R.E., Consultant, South-Eastern Regional Radiotherapy Service, Royal Infirmary and Western General Hospital, Edinburgh, Scotland.

32. Harry Z. Mellins, M.D., Professor and Chairman, Department of Radiology, State University of New York, Brooklyn, New York.

33. Jarrell E. Miller, M.D., Director of Radiology, Baylor University Hospital, Dallas, Texas.

34. Lowell S. Miller, M.D., Associate Radiotherapist, Department of Radiology, M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, Texas.

35. William T. Moss, M.D., Director, Therapeutic Radiology, Chicago Wesley Memorial Hospital, Chicago, Illinois.

36. Walter T. Murphy, M.D., Director of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo, New York.

37. Sidney W. Nelson, M.D., Professor and Chairman, Department of Radiology, The Ohio State University, Columbus, Ohio.

38. Charles M. Nice, Jr., M.D., Professor and Head of Department of Radiology, Tulane University, New Orleans, Louisiana.

39. F. W. O'Brien, Jr., M.D., Department of Radiation Therapy, Cleveland Clinic, Cleveland, Ohio.

40. Robert S. Ormond, M.D., Department of Radiology, Henry Ford Hospital, Detroit, Michigan.

41. Bernard Roswit, M.D., Chief, Radiotherapy Service, Veterans Administration Hospital, Bronx, New York.

42. Philip Rubin, M.D., Chief, Division of Radiotherapy and Associate Professor of Radiology, University of Rochester, School of Medicine, Rochester, New York.

43. Herbert M. Stauffer, M.D., Professor and Director, Diagnostic Division, Department of Radiology, Temple University Hospital, Philadelphia, Pennsylvania.

44. John P. Storaasli, M.D., Department of Radiol ogy, University Hospitals of Cleveland, Cleveland, Ohio.

45. Juan M. Taveras, M.D., Department of Radiology, Neurological Institute, New York, New York.

46. D. H. Thomson, M.B., Ch.B., D.M.R.T., Radiotherapist, The Ontario Cancer Foundation, London Clinic, Victoria Hospital, London, Ontario, Canada.

47. Sidney P. Traub, M.D., Associate Professor of Radiology, University Hospital, University of Saskatchewan, Saskatoon, Saskatchewan, Canada.

48. William J. Tuddenham, M.D., Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

49. T. A. Watson, M.B., Ch.B., D.M.R., Professor of Therapeutic Radiology and Director of Cancer Services for Saskatchewan, University of Saskatchewan Medical School, Saskatoon, Saskatchewan, Canada.

50. H. Stephen Weens, M.D., Emory University, School of Medicine, Atlanta, Georgia.

51. Robert E. Wise, M.D., Department of Radiology, The Lahey Clinic, and New England Baptist Hospital, Boston, Massachusetts.

52. George T. Wohl, M.D., Associate Professor of Radiology, Temple University School of Medicine; Chief, Radiology Service, Veterans Administration Hospital, Philadelphia, Pennsylvania.

DESCRIPTION OF COURSES

COURSE 101
and
COURSE 201

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-urinary Tract of Infants and Children

Course 101. Probably the most important examination in Pediatric Radiology with the highest yield of positive returns is intravenous urography, and yet, in spite of its importance, it is a study that is frequently poorly executed. There are a multitude of reasons for this. The speaker will attempt to show his own problems with this procedure and how, by working at it over a long period of time, he arrived at a technique which nine out of ten times results in a diagnostic study. This technique will include various methods of reducing the roentgen exposure to the gonads. It will also illustrate the importance of obtaining a voiding urethrogram in the male as one of the final steps in the procedure.

Having presented the technique of intravenous urography, the speaker will then attempt to illustrate his own evolutionary understanding of posterior-urethral valves, ectopic ureteroceles, absence of the abdominal musculature, and of as many other congenital anomalies as time will permit.

Mediastinal Mass Lesions in Infants and Children

Course 201. Mass lesions occurring in the mediastinum can be diagnosed with a high degree of accuracy simply by knowing whether they occur in the front, in the middle or in the back. This course will attempt to give a comprehensive discussion of most of the mass lesions which occur in the mediastinum. There are a few differences between the younger age group and the older age group and these differences will be discussed.

COURSE 102

Tuesday

FELIX G. FLEISCHNER, M.D.
Boston, Massachusetts

Pulmonary Embolism and Acute Cor Pulmonale

Pulmonary embolism is a disease frequently observed in a general hospital. Our knowledge of the pathogenesis, morphology, and clinical diagnosis, including the roentgen diagnosis, is of very recent date and by no means a closed chapter. With constant reference to the background of pathophysiology and morphology and to the clinical constellation, the roentgen diagnosis of pulmonary embolism with infarct formation, based on the work of Hampton and Castleman (Am. J. Roentgenol. & Rad. Therapy, 1940, *46*, 305) will be thoroughly discussed and illustrated. Furthermore, emphasis will be placed on the roentgen diagnosis of pulmonary embolism without infarct formation and on the understanding and recognition of the syndrome of acute cor pulmonale (Fleischner, *Canad. M. A. J.*, 1958, *78*, 653).

COURSE 103
and
COURSE 203

Tuesday and Wednesday

ALEXANDER STEWART MACMILLAN, M.D.
Boston, Massachusetts

Sinuses and Mastoids, Lateral Neck and Larynx

Course 103. Sinuses.
Course 203. Mastoids, Lateral Neck and Larynx.

COURSE 104

Tuesday

GEORGE JACOBSON, M.D.
Los Angeles, California

Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint

The Atlanto-Axial Joint possesses unique features of structure and function, which make it subject to certain rather distinctive injuries. Many of these can be demonstrated by rather simple examination while others require more elaborate techniques.

The following will be discussed:

1. Normal anatomy and embryology.
2. Normal motion of the Atlanto-Axial Joint.
3. Methods of examination.
4. Readily recognizable fractures and dislocations will be dealt with briefly.
5. A major portion of the period will be devoted to a discussion of injuries which are difficult to demonstrate and somewhat unusual.

COURSE 105
and
COURSE 205

Tuesday and Wednesday

ARNOLD L. BACHMAN, M.D.
New York, New York

Roentgenographic Diagnosis of the Larynx and Pharynx

The following are discussed in detail in two sessions: Roentgenographic techniques for examination

of the soft tissues of the neck; roentgenographic normal anatomy and physiology of the larynx; laryngeal paralysis; roentgenology of normal and abnormal swallowing mechanisms; neoplasms of the larynx and pharynx; and extension of the laryngopharyngeal tumors.

Also to be discussed are foreign bodies of the cervical esophagus, laryngocele, cricopharyngeal spasm and roentgenology of the adenoids.

COURSE 106
and
COURSE 206

Tuesday and Wednesday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy, physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 106. (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 206. (1) Anatomic variations in the pulmonary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 107

Tuesday

HERBERT M. STAUFFER, M.D.
Philadelphia, Pennsylvania
JOHN KIRKPATRICK, M.D.
Philadelphia, Pennsylvania

Image Intensification and Cinefluorography

Experience with several types of image intensifiers will be described. Present applications, including use of closed-circuit television, in research and practice will be discussed and illustrated. Limitations of present devices will be considered. Since present instruments are particularly suited to pediatric practice, this aspect will receive special consideration.

COURSE 108
and
COURSE 208

Tuesday and Wednesday

EMANUEL J. LEVIN, M.D.
Brooklyn, New York

The Fundamentals of Bone Disease

There are characteristic radiologic patterns produced by diseases of the bones which permit a definite and accurate diagnosis. The reactions in the soft tissues, periosteum and cartilage are often of greater differential value than the changes in bone. Furthermore, many osseous diseases have peculiarities of destruction, production and localization which in themselves are of diagnostic significance. The detection and correct assessment of the varied roentgen patterns will permit an accurate diagnosis in a high percentage of cases.

An analytic approach to the differential diagnosis of diseases of the bones will be presented with emphasis placed on fundamental aspects.

I. Explanation and description of terms
   A. Soft tissue reactions
   B. Periosteal reactions
   C. Osseous reactions
II. Tumors
   A. Benign
   B. Malignant
III. Infections
   A. Acute
   B. Chronic

COURSE 109
and
COURSE 209

Tuesday and Wednesday

GEORGE T. WOHL, M.D.
Philadelphia, Pennsylvania
ERNEST AEGERTER, M.D.
Philadelphia, Pennsylvania

Unusual Problems in Bone Diagnosis

During the past four years a regular monthly meeting has been held in Philadelphia known as the Orthopedic-Radiologic-Pathologic Conference. At this conference, problems of unusual interest in the field of bone pathology are presented by all of the major teaching institutions in the Philadelphia area and discussed from the standpoint of their importance to these three specialties. A remarkable collection of unusual bone cases has been gathered which is of much teaching value. Selected cases from the conference files considered to have direct important clinical application will be presented and discussed from the standpoint of roentgen diagnostic characteristics and pathologic features. While many of the cases will represent benign and malignant bone tumors, other bone problems of particular roentgen interest will also be included.

COURSE 110

Tuesday

HARRY Z. MELLINS, M.D.
Brooklyn, New York

Nonobstructive Acute Abdominal Disorders

An analytic approach to the roentgenographic diagnosis of the nonobstructive acute abdominal disorders will be presented. Emphasis will be placed upon the plain film findings. The importance of serial or follow-up examinations will be stressed. The course will include inflammatory conditions, trauma, toxic conditions and vascular disorders.

COURSE 121

Tuesday

M. Y. COLBY, JR., M.D.
Rochester, Minnesota

Radiation Therapy of Pituitary Tumors

This course will consist of a review of the experience at the Mayo Clinic in the treatment of pituitary tumors by radiation therapy. Indications for treatment, treatment techniques and the problem of evaluating results will be discussed.

COURSE 122

Tuesday

JOHN P. STORAASLI, M.D.
Cleveland, Ohio

Radioactive Isotopes—Therapeutic Application

A survey will be made of the more important therapeutic applications of radioisotopes. To be included will be a discussion of the rationale of therapy and a comparison of other methods of treatment. The isotopes to be covered in detail will be radioactive phosphorus in the treatment of polycythemia, leukemia, and metastatic breast carcinoma in bone; radioactive iodine for diseases of the thyroid gland and euthyroid cardiacs; radioactive colloidals for malignant serous effusions; and radioactive strontium for beta-ray therapy of ocular lesions.

COURSE 123

Tuesday

MANUEL GARCIA, M.D.
New Orleans, Louisiana

The Treatment of Carcinoma of the Cervix

The techniques and dosimetry of roentgen-ray and radium therapy for carcinoma of the cervix will be discussed. Modifications in the plan of treatment dictated by features of the tumor and of the host will be described. Special emphasis will be placed on time dose relations and the volume factor. The prognostic influence of all these factors will be demonstrated by the tabulation of end results

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COURSE 124

Tuesday

J. ROBERT ANDREWS, M.D.
Bethesda, Maryland

The Modification of the Radiation Response and the Relief of Cancer

The course will begin with a review of the historical, intuitive, and rational bases for modification of the radiation response. Experimental biologic evidence for modification will be presented and mechanisms will be discussed. Attention will be given to dose-survival curves for cell cultures of various types and the interpretation of such curves. Various aspects of the experimental clinical approach to modification of the radiation response in cancer therapy will be considered with special attention being given to the design and to the mounting of cooperative joint clinical studies and to problems of human experimental pharmacology.

COURSE 202
and
COURSE 302

Wednesday and Thursday

RICHARD G. LESTER, M.D.
Minneapolis, Minnesota

Roentgen Diagnosis of Operable Cardiac Lesions

Corrective surgery for most of the cardiac lesions of childhood has become a reality. In addition, palliative procedures for a number of other congenital conditions, for rheumatic valvular heart disease of most types and for certain acquired heart diseases have radically changed the prognosis for many patients with heart disease. As a result, the necessity for precise diagnosis has been brought into focus. In Course 202 the place of roentgenology in the clinical examination of patients with heart disease will be outlined, with special emphasis on those conditions in which the possibility of correction or surgical amelioration exists. Interpretation in terms of pathologic anatomy and physiology will be stressed.

Course 302 will review in detail the contribution of the conventional roentgen examinations to the diagnosis of these conditions. A method of analysis of the dynamic and anatomic features revealed by these techniques will be presented. Correlation of the roentgen findings with the data provided by other conventional forms of clinical examination will be stressed.

COURSE 204

Wednesday

ROBERT E. WISE, M.D.
Boston, Massachusetts

Intravenous Cholangiography

Intravenous cholangiography retains its important position in the armamentarium of the radiologist concerned with diagnostic problems of the biliary tract. Data presented in the course have been gleaned from six years' experience with approximately 2,500 injections. Two thousand and thirty-two cases have been subjected to statistical analysis. Roentgenologic preoperative diagnoses have been correlated with operative findings in 466 cases, of which 225 were in the postcholecystectomy state.

Technique and interpretation will be considered in detail with emphasis on the postcholecystectomy patient as related to the time-density retention concept. Pitfalls have been analyzed and will be presented. Specific findings secondary to calculi, tumors, strictures, fibrosis of the sphincter of Oddi, pancreatitis and so forth have been analyzed for presentation.

The great value of the procedure lies in the accuracy with which organic biliary tract disease can be confirmed or excluded in the postcholecystectomy patient. Although the accurate diagnosis of a pathologic state may lead to surgical relief, at least of equal value is the exclusion of unnecessary surgical exploration.

COURSE 207
and
COURSE 307

Wednesday and Thursday

JARRELL E. MILLER, M.D.
Dallas, Texas

Roentgen Examination of the Stomach and Small Bowel

The roentgen examination of the stomach and small bowel is the one area where the radiologist can and must remain supreme. Many specialists, whose specialty exists as we know it today because of the roentgen examination, become quite skilled in interpreting films in their field. The really good physicians in these specialties will not fail to utilize the services of a radiologist who has himself acquired requisite skill in the interpretation of films within their field. Not all radiologists are able to maintain adequate skill in interpretation of special studies such as cerebral angiograms, pneumograms, angiocardiograms and other special procedures when they have little or no opportunity to see them. The upper gastrointestinal tract study is one of the most frequently performed examinations where the radiologist plays a dominant role. It is necessary, therefore, that he acquire and maintain a superior skill in performing these examinations as well as interpreting the results. He must be prepared to interpret films made elsewhere as well as to influence the quality of the examinations that are done elsewhere.

The first course will deal principally with technique of the upper gastrointestinal tract examination as well as the small bowel study. The minimum film requirement for an adequate upper gastrointestinal tract study will be demonstrated. Techniques for combination procedures which can be done without compromising any of the examinations will also be shown. Pitfalls and errors in interpretation will be discussed along with methods of avoiding them.

COURSE 210

Wednesday

HAROLD G. JACOBSON, M.D.
New York, New York

The Vaterian Segment: Normal and Abnormal

The roentgen anatomy of the normal vaterian structures *in vitro* will be described utilizing cadaver material. The *in vivo* appearance of the normal vaterian papillae in the upper gastrointestinal tract examination will be illustrated and emphasized after a brief description of the various methods of the roentgen examination of the vaterian region. There will then follow the discussion of some of the important disease entities involving the vaterian area. The section on abnormalities will be dealt with against the background of a classification of enlargement of the papilla of Vater divided into two major categories: centric and eccentric enlargements. The centric enlargements will include those associated with common duct stone, acute and chronic relapsing pancreatitis, and carcinoma of the papilla and ampulla of Vater. The eccentric enlargements will be demonstrated mainly by cases with peptic ulcer disease in the proximal duodenum. The differential diagnosis of the various disease states causing these enlargements will be emphasized and illustrated with the aid of an outline long in use for evaluating abnormalities of the vaterian segment. The roentgen findings in carcinoma of the head of the pancreas will also be briefly described, particularly as these changes relate to the vaterian segment.

COURSE 221

Wednesday

JEAN BOUCHARD, M.D.
Montreal, Quebec, Canada

Role of Radiation Therapy in the Treatment of Primary Intracranial Neoplasms

Personal experience in the use of ionizing radiation in the management of neoplasms involving the central nervous system is based upon a series of 836 pa-

tients treated over a twenty year period between 1939 and 1958.

An analysis of the results of irradiation, used in combination with surgery or alone, will be presented for each type of primary intracranial neoplasm adequately irradiated between 1939 and 1953 inclusive (399 cases). The quality or degree of useful functional survival will be stressed in relation to the length of survival. Special reference will be made to our experience in children who have been irradiated for intracranial neoplasms, the ages ranging from six months to fifteen years. A very brief reference will be made concerning the irradiation of selected cases of brain metastases. Pituitary tumors will not be included in this presentation.

Discussion of the indications for treatment, technique, reactions, radiation hazard and other aspects of the problem will be presented.

COURSE 222

Wednesday

J. C. F. MACDONALD, M.A., Ph.D.
London, Ontario, Canada
D. H. THOMSON, M.B., Ch.B., D.M.R.T.
London, Ontario, Canada

Evolution in Cobalt Teletherapy Techniques

In this course the development of techniques in cobalt 60 beam therapy over a period of nine years will be presented. Fixed field, wedge field, arc and full rotation techniques will be covered in detail and two alternate methods of dose estimation will be discussed.

COURSE 223

Wednesday

LOWELL S. MILLER, M.D.
Houston, Texas

Supervoltage Irradiation in Carcinoma of the Bladder

Results of various methods of treatment of bladder cancers will be surveyed, with the emphasis on external supervoltage irradiation.

Clinical staging will be reviewed, practical techniques for localization and irradiation discussed, and what the urologist and radiotherapist may expect from these techniques, surveyed in terms of complications.

Clinical management of the individual patient before, during, and after irradiation will be outlined in the discussion.

COURSE 224

Wednesday

BERNARD ROSWIT, M.D.
Bronx, New York

Cancer Chemotherapy in Radiologic Practice

Patients and colleagues generally look to the therapeutic radiologist as the most knowledgeable specialist in the treatment of inoperable cancer. Cytotoxins, antimetabolites, antibiotics, radiation synergists and steroids have recently gained great importance as adjuncts to radiation treatment. It is essential that radiologists keep abreast of these new developments to improve their management of patients with advanced disease. This course has therefore been planned by a radiologist for radiologists, to discuss available drugs, dosage, indications, clinical effectiveness, complications, and integration with the radiation treatment. The trend of future developments will be reviewed.

COURSE 301

Thursday

CHARLES M. NICE, JR., M.D.
New Orleans, Louisiana

Roentgen Manifestations of Collagen Diseases in the Chest

A series of 109 patients with five collagen diseases admitted to the University of Minnesota Hospitals during the past fifteen years has been reviewed. Roentgenograms of the chest probably did not contribute to the diagnosis at any time in about one-third of the patients. In another one-third, one or two abnormalities appeared in the chest at some phase of the disease process, serving as confirmatory findings which could be considered of help in following the progress of the patient. Of special interest to the roentgenologist, however, is the final one-third of the patients in whom a combination or sequence of roentgen findings should actually lead one to suspect a collagen disorder. If there are clinical and laboratory findings suggesting the presence of a collagen disease, these roentgenographic changes will aid in the differentiation of the various types in about one-fifth of the patients.

There are many roentgen findings in the chest which appear in various collagen disorders, most of which are nonspecific. A nonspecific type of cardiac enlargement, interstitial pneumonitis and pleural effusion are signs which may be seen in any of the collagen diseases either individually or in combination. Combinations of these and other signs, such as hilar vascular enlargement, small nodules in the lungs, interstitial pulmonary fibrosis and other less commonly observed signs, may be of great help in actually diagnosing or following the progress of the condition or in evaluating the response to therapy.

COURSE 303
and
COURSE 403

Thursday and Friday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

Course 303. 1. Technique, including a discussion of the various barium preparations. 2. Regional enteritis. 3. The malabsorption syndrome, including sprue, lymphosarcoma and Whipple's disease. 4. Problems in the interpretation of functional changes.

The Small Intestine and Duodenum

Course 403. A. 1. Infarction of the small bowel. 2. Tumors. 3. Miscellaneous disorders, including scleroderma, amyloidosis, lupus erythematosus, allergy.

B. This will include a discussion of lesions of the duodenal bulb excluding duodenal ulcers.

COURSE 304
and
COURSE 404

Thursday and Friday

JOHN A. CAMPBELL, M.D.
Indianapolis, Indiana

Radiology of the Urinary Tract

Interpretation of roentgen studies of the urinary tract requires an understanding of the basic embryologic, anatomic, physiologic, and pathologic factors involved. In addition, the indications, limitations, and specific usefulness of the various methods of roentgenologic investigation of the urinary tract structures should be fully appreciated. Illustrative case material will be presented to demonstrate the application of significant roentgen signs to the differential diagnosis of certain urinary tract problems encountered by the roentgenologist.

Course 304. (1) Technical aspects, (2) anomalies, variants, and trauma, and (3) calculi and infections.

Course 404. (1) Renal and perirenal mass lesions, (2) vascular conditions, and (3) obstructions and dysfunctions.

COURSE 305
and
COURSE 405

Thursday and Friday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses

Course 305. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and various skull types. Analysis of individual bone components in producing the various lines and shadows seen in plain roentgenograms of the skull. This analysis will be applied to a study of the sphenoid, frontal, ethmoid, parietal and occipital bones in various projections.

Course 405. Detailed roentgen anatomic analysis of the paranasal sinuses and mastoids with description of roentgenographic techniques employed. Study of the roentgen features of the middle cranial fossa and their relation to the sphenoid sinuses. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. In conclusion, the maxillofacial components of the cranium will be examined, including the lacrimal canals and soft tissue delineation.

COURSE 306

Thursday

GISÈLE LUSSIER, M.D.
Montreal, Quebec, Canada

The Roentgen Manifestations of the Peripheral Nervous System Tumors

In this course, the various aspects of these tumors and the numerous sites where they occur will be illustrated with the aid of slides. The subject will be divided in three parts: (1) the tumors of the peripheral nerves, (2) the tumors of the sympathetic ganglia, and (3) the tumors of the paraganglia. All the cases which will be presented have been selected from the material of the Radiologic Pathology Department of the Armed Forces Institute of Pathology, Washington, D. C.

COURSE 308

Thursday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

The inadequacy of "density," "resolution," "contrast" and "unsharpness" as criteria for evaluating the "quality" of diagnostic roentgenograms will be discussed, and new criteria of "film quality" will be presented.

The significance of the various physical factors affecting the "quality" of roentgenograms will be considered, and the indications for so-called "high-kilovoltage" and "supervoltage" radiography as well as for the use of the "micro-focus" roentgen-ray tube will be specifically noted.

Recent observations concerning view-box and surrounding illumination, the control of glare, the choice of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 309

Thursday

H. STEPHEN WEENS, M.D.
Atlanta, Georgia
BRIT B. GAY, JR., M.D.
Atlanta, Georgia

Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes

I. Methods of roentgenologic study of pulmonary circulation: (a) roentgenoscopy with image amplification, (b) routine roentgenograms, (c) tomograms, (d) pulmonary angiograms, and (e) cineroentgenography.

II. Basic function of pulmonary circulation; normal pulmonary arterial and venous pressure; normal pulmonary blood flow.

III. Thromboembolism in pulmonary circulation.

IV. Increased pulmonary blood flow. Definition. Etiology: left to right shunts; admixture lesions. Roentgenologic signs. Pulsations of the pulmonary arteries in increased pulmonary blood flow.

V. Decreased pulmonary blood flow. Definition. Etiology: right to left shunts with pulmonary stenosis of atresia. Roentgenologic signs. Roentgenologic appearance of collateral circulation to the lungs through bronchial arteries or anomalous arteries. Pulsations of pulmonary arteries in decreased pulmonary blood flow. Special consideration of pulmonary vascular changes and pulsations in isolated pulmonary valvular stenosis.

VI. Pulmonary venous hypertension—pulmonary edema. Roentgenologic signs. Significance of Kerley's (septal) lines. Special consideration of pulmonary venous changes in mitral stenosis. Importance of minimal signs of pulmonary interstitial edema as an indication of acute myocardial infarction.

VII. Pulmonary arterial hypertension. Definition. Etiology: roentgenologic signs. Pulsations of pulmonary arteries in pulmonary hypertension.

COURSE 321

Thursday

CHARLES L. MARTIN, M.D.
Dallas, Texas

Low Intensity Radium Needles in the Treatment of Cancer

Low intensity radium needles still constitute one of the most efficient and convenient mediums available for the treatment of accessible carcinomas with interstitial irradiation. Techniques and dosage calculations will be described; the results obtained in cancer of the face, nose, ear, oropharynx, tongue, floor of the mouth, gum, cheek, palate, metastatic cervical lymph nodes and the cervix will be illustrated with lantern slides.

COURSE 322

Thursday

WALTER T. MURPHY, M.D.
Buffalo, New York

Radiation Therapy of Skin Cancer

The course will illustrate clinical results of various techniques currently used at the Roswell Park Memorial Institute for small and large lesions involving various anatomic sites, with and without invasion of cartilage and bone. The discussion will emphasize: (1) the advantages of small increment fractionation as compared to the so-called "massive dose" technique; (2) the proper selection of radiation quality (half value layer 0.06 mm. Al, 30 kv. peak up to half value layer 0.9 mm. Cu 200 kv. peak) and the indications for combining different radiation qualities; (3) the influence of the cancer site upon the selection of field size; (4) the care of the patient during and following irradiation; and (5) the factors influencing complications and sequelae.

COURSE 323
and
COURSE 423

Thursday and Friday

PHILIP RUBIN, M.D.
Rochester, New York
and
GEORGE CASARETT, Ph.D.
Rochester, New York

Clinical Radiopathology for the Practicing Radiologist

Concepts of radiation changes and injuries continue to evolve as clinical experience unfolds and animal experimentation increases. The integration of the principles of radiation therapy as practiced clinically and of radiobiology as observed in animals is an ever present challenge for the most avid reader. The limitations of establishing clinical tolerance for specific organs in varied series of patients and the possible errors in extrapolation from laboratory mammals to man represent the two horns of the dilemma.

A review of major organ systems will be undertaken with presentation of information in a logical sequence. The histophysiology of the organ and the radiopathologic changes will be analyzed as a basis for understanding the clinical course of induced radiation injury. Emphasis will be placed on clinical symptoms and signs and physical and biologic factors evoking this reaction to therapy. Threshold doses will be discussed. Case presentations with pertinent radiographic and laboratory data will be offered for discussion. A highly selected bibliography of key references for the general radiologist practicing radiotherapy will be offered. (A more detailed syllabus of our course, "Clinical Radiopathology," for resident physicians and for those responsible for teaching a course to a house staff will be available on request.)

COURSE 324

Thursday

MAURICE LENZ, M.D.
New York, New York

The Fight Against Cancer in the U.S.S.R.

During a twenty-four day visit to the U.S.S.R. as an Exchange Lecturer for our State Department in October, 1959, I had the opportunity to observe the fight against cancer in the U.S.S.R. This is directed by the Ministry of Health in Moscow with its subsidiary, yet independent, Ministries in each of the 15 republics composing the U.S.S.R. They are responsible for the cancer education of the medical profession and public, and for the prophylaxis and treatment of cancer; they function through a network of cancer hospitals, institutes, dispensaries, smaller clinics and cancer detection clinics.

Prophylaxis and treatment are standardized, so that a specific type of cancer is supposed to be treated by similar surgery, radiotherapy or chemotherapy, whether the patient is seen in an outlying city or in Moscow. The method of attack in individual cancers is specified, according to the anatomic site, clinical stage and microscopic character of the cancer, in a handbook available in the Oncologic Services. If the physician is unable to follow this advice, he refers the patient to the doctor or institution qualified to carry out the treatment. In spite of some disadvantages, such standardization permits rapid accumulation of a great number of similar cancers treated by similar methods, thus facilitating their statistical evaluation. Medical care is free in the U.S.S.R. and early diagnosis is encouraged by constant propaganda to the public and medical profession.

COURSE 325

Thursday

T. A. WATSON, M.B., Ch.B., D.M.R.
Saskatoon, Saskatchewan, Canada

Advantage of Supervoltage Radiation in Treatment of Cancer of the Cervix, Uterus and Ovary

The particular technical advantages of this technique used alone, or in conjunction with intracavitary radium, or following surgery will be discussed.

COURSE 401

Friday

F. W. O'BRIEN, JR., M.D.
Cleveland, Ohio

Cysts and Tumors of the Mandible

The embryologic development of the mandible will be briefly reviewed.

The differential diagnosis of mandibular lesions is made difficult by the presence of cysts or tumors of dental origin. Roentgenographic and histologic correlation is often lacking. An attempt will be made to classify these lesions as to etiology, pathology and roentgen findings.

Examples of squamous, radicular, dentigerous and traumatic cysts will be shown. Also included are the roentgenographic features of giant cell tumors, adamantinomas, odontomas, fibrous dysplasia, myelomas and some malignant neoplasms. Differential diagnostic features will be discussed.

COURSE 402

Friday

SIDNEY P. TRAUB, M.D.
Saskatoon, Saskatchewan, Canada

Myelography—Technique and Interpretation

This course will stress the practical aspects of myelography of interest and importance to the roentgenologist. The following outline will be followed:

1. Brief historical review.
2. Indications for myelography; contraindications.
3. Pertinent roentgenologic anatomy and anatomic variations.
4. Basic principles in myelographic technique.
5. Complications of myelography and their significance.
6. Lumbar intervertebral disk protrusions: (a) the value of preliminary chest and spine roentgenograms; (b) obstructing and partially obstructing disk protrusions; (c) posterolateral disk protrusions; (d) laterally situated disk protrusions; (e) significance of minor myelographic deformities in the presence of lumbar disk protrusions.
7. The postoperative lumbar myelogram.
8. Cervical and thoracic disk protrusions.
9. Summary and general discussion including: (a) common errors in myelographic interpretation; (b) value and limitations of myelography in patients with suspected intervertebral disk protrusions.

COURSE 406

Friday

WILLIAM R. EYLER, M.D.
Detroit, Michigan
ROBERT S. ORMOND, M.D.
Detroit, Michigan

The Acute Abdomen

This course is designed primarily to review the roentgenologic manifestations of acute abdominal disease, particularly, as manifested on plain abdominal films. When appropriate for illustrative purposes, associated examinations with contrast materials will be presented. The following outline includes the subjects to be discussed:

1. Anatomy of the peritoneal cavity as shown by pneumoperitoneum.
2. Differentiation of the hollow viscera.
3. Vascular Diseases: (a) aneurysms, dissecting and arteriosclerotic; (b) occlusive vascular disease; (c) blood dyscrasias; (d) polyserositis; (e) collagen diseases.
4. Inflammatory diseases of hollow viscera: (a) acute appendicitis, acute cholecystitis, gastritis, enteritis and cholecystitis; (b) regional enteritis; (c) tuberculosis; (d) generalized peritonitis; (e) localized abscesses.
5. Inflammatory diseases of solid viscera: (a) acute pancreatitis; (b) acute perinephric and obstructive urinary tract diseases.
6. Mechanical obstructions of the digestive tube: (a) neoplastic diseases; (b) extrinsic pressure; (c) special mechanical obstructions including intussusception, volvulus, hernia and band.
7. Traumatic conditions: (a) solid viscera; (b) hollow viscera.
8. Miscellaneous conditions causing or simulating the acute abdomen including sprue, neurologic disorders, toxic agents and endocrine malfunctions.

COURSE 407

Friday

JUAN M. TAVERAS, M.D.
New York, New York

Anatomy of the Subarachnoid Cisterns in Relation to the Diagnosis of Tumors of the Posterior Fossa

In this instructional course the anatomy of the cisterns of the posterior fossa will be described in as much detail as time permits and examples of pathologic entities affecting the various cisterns will be presented.

COURSE 408

Friday

SIDNEY W. NELSON, M.D.
Columbus, Ohio

Bronchography

The radiologist who can properly perform and interpret bronchograms finds himself in a position where he can make astonishing contributions to the diagnosis, and understanding of the physiopathology of many chest diseases. The proper therapy for any disease which affects the bronchial tree is quite dependent on the accurate determination of the extent of the disease as shown on a complete bronchographic study. In this instructional course, therefore, emphasis will be placed on the description of the technique of bronchography, including the method of anesthesia, choice of contrast materials, and spot filming. The making of spot films during the period of optimum filling of the main bronchopulmonary segments will be stressed and illustrated.

Normal anatomy and some of the common anatomic variations of importance in roentgenographic interpretation will be discussed and illustrated. It will be shown that a careful and systematic identification of each of the major segmental bronchi is important, because a segmental bronchus completely occluded at its orifice often cannot be identified,

but may be confused with an adjacent segmental bronchus which has become relocated due to atelectasis distal to the completely obstructed unidentified bronchus. The bronchographic manifestations of chronic bronchitis, emphysema, congenital polycystic lung, and minimal degrees of bronchiectasis will also be described and illustrated.

It is hoped that this presentation will encourage radiologists to perform bronchograms more frequently for their nonradiologist colleagues. The technique can be mastered with a little effort and with extremely few exceptions, the bronchographic technique to be described can be quickly performed by the radiologist and his technical staff, precluding the necessity for resorting to the "team" approach which is often so time consuming and frustrating in large busy hospitals.

COURSE 409

Friday

NATHANIEL FINBY, M.D.
New York, New York

Male Cystourethrography

A general survey of male cystourethrography will stress normal and pathologic roentgenographic anatomy. Congenital and acquired diseases of the lower urinary tract will be demonstrated on cystourethrograms.

COURSE 410

Friday

ROBERT L. EGAN, M.D.
Houston, Texas
GERALD D. DODD, M.D.
Houston, Texas

Mammography

Over four years' experience has been gained with soft tissue roentgenography of the breast in a tumor institute with an active breast service. These studies have proved a highly accurate and valuable adjunct to the clinical and pathologic evaluation of breast lesions.

A simple and readily adaptable roentgenographic technique will be presented. Cases illustrative of typical benign and malignant disease will be shown and the diagnostic features of each described and tabulated. Differential diagnosis, sources of error, limitations of the procedure and the clinical and pathologic implications of the method will be discussed.

The results of a statistical analysis of the first 1,000 consecutive examinations (all with a follow-up from one to four years) will be reviewed. The error in 245 cases of malignant disease was 0.82 per cent.

COURSE 421

Friday

RUTH GUTTMANN, M.D.
New York, New York

The Management and Results in the Treatment of Inoperable Carcinoma of the Lung with Two Million Volt or Cobalt 60 Therapy as Compared to the Results with Orthovoltage or One Million Volt Therapy

One-hundred and fifty-three cases of advanced inoperable carcinoma of the lung, with spread to adjacent organs and/or distant lymph node bearing areas, will be surveyed. All of the patients were treated with supervoltage therapy, most of them with a two million volt roentgen-ray unit, some with a cobalt 60 unit.

The treatment approach, including single and total dosage, and a comparison of the advantages and disadvantages of these two supervoltage units in the treatment of carcinoma of the lung will be presented. This demonstration will show treatment plans on the 2 mv. and cobalt units (1) through stationary opposing fields, (2) with full rotation and (3) with scanning. A discussion of the tolerance to therapy, of subjective symptomatic improvement and of the objective findings will follow. Special emphasis will be placed on film demonstrations before and after therapy as well as on a comparison of the response of various malignant lesions of the lung to irradiation as proved by microscopic examination. The ultimate results as far as survival time is concerned will be discussed and compared with a second similar group of patients who were treated with 250 kv. therapy and a third group of patients who were treated with one million volt irradiation. Detailed tables of this material will be used to illustrate the pertinent points.

COURSE 422

Friday

WILLIAM T. MOSS, M.D.
Chicago, Illinois

Radiotherapy of Carcinoma of the Breast —One Response to a Therapeutic Dilemma

A discussion will be presented of the pros and cons of preoperative and postoperative irradiation. A rationale for the care of these patients will be developed and our technique of irradiation described.

COURSE 424

Friday

JUAN A. DEL REGATO, M.D.
Colorado Springs, Colorado

Leukemias, Lymphomas, and Hodgkin's Disease

In the treatment of this group of diseases, it is important first to recognize as much as possible their individual identities, for the prognosis varies and the aggressivity of the therapist may, of necessity, be different.

The course will present a discussion of the clinical handling of these problems and of the expected results of radiotherapy.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 27, 1960**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
101—Hope	Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-Urinary Tract of Infants and Children	**121—Colby**	Radiation Therapy of Pituitary Gland Tumors
102—Fleischner	Pulmonary Embolism and Acute Cor Pulmonale	**122—Storaasli**	Radioactive Isotopes—Therapeutic Application
103—Macmillan	Sinuses		
104—Jacobson	Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint	**123—Garcia**	The Treatment of Carcinoma of the Cervix
105—Bachman	Roentgenographic Diagnosis of the Larynx and Pharynx		
106—Felson	Some Fundamentals in Chest Roentgen Interpretation	**124—Andrews**	The Modification of the Radiation Response and the Relief of Cancer
107—Stauffer Kirkpatrick	Image Intensification and Cinefluorography		
108—Levin	The Fundamentals of Bone Disease		
109—Wohl Aegerter	Unusual Problems in Bone Diagnosis		
110—Mellins	Nonobstructive Acute Abdominal Disorders		

SECTION ON INSTRUCTION
ORDER SHEET

It is important to register for the Instruction Courses as early as possible since the number admitted to each course will be limited. *It is also very important that first, second and third choices be listed for each period.* All orders for tickets will be filled according to the postmark on the envelopes.

Non-members of the American Roentgen Ray Society, except full-time graduate students in Radiology, will pay $2.00 for each course-period or a maximum of $8.00 for 4 courses. *Non-members' fees must accompany this order sheet and will not be returned unless cancellation is received before September 20, 1960.*

Tickets for courses will be mailed to each registrant whose order reaches the office of the Director on or before September 17, 1960. They will be sent to the address below unless registrant directs otherwise.

Please fill out the following (type or print):

Check ☐ Member ☐ Guest ☐ Graduate Student in Radiology at:

.....
Last Name First Name or Initials

.....
Street Address

....
City Zone State

Those whose advance registration reaches the office of the Director after September 17, 1960, may obtain their tickets at the Registration Desk of the Section on Instruction on Monday afternoon, September 26, 1960 and thereafter. At this time tickets may also be obtained for courses not sold out.

For convenience in selecting your courses consult the condensed schedule of courses.

BE SURE TO FILL OUT SECOND AND THIRD CHOICES FOR EACH PERIOD

Period	First Choice		Second Choice		Third Choice	
	Course No.	Instructor	Course No.	Instructor	Course No.	Instructor
Tuesday						
Wednesday						
Thursday						
Friday						

Previous to September 17, 1960, send this order sheet to:

DR. HAROLD O. PETERSON, 1995 WEST COUNTY ROAD "B," ST. PAUL 13, MINNESOTA.

After September 17, 1960, mail to:

DR. HAROLD O. PETERSON, American Roentgen Ray Society, Ambassador Hotel, Atlantic City, New Jersey.

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.*** **September 28, 1960**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
201—Hope	Mediastinal Mass Lesions in Infants and Children	**221—Bouchard**	Role of Radiation Therapy in the Treatment of Primary Intracranial Neoplasms
202—Lester	Roentgen Diagnosis of Operable Cardiac Lesions		
203—Macmillan	Mastoids, Lateral Neck and Larynx	**222—MacDonald Thomson**	Evolution in Cobalt Teletherapy Techniques
204—Wise	Intravenous Cholangiography		
205—Bachman	Roentgenographic Diagnosis of the Larynx and Pharynx	**223—Miller**	Supervoltage Irradiation in Carcinoma of the Bladder
206—Felson	Some Fundamentals in Chest Roentgen Interpretation		
207—Miller	Roentgen Examination of the Stomach and Small Bowel	**224—Roswit**	Cancer Chemotherapy in Radiologic Practice
208—Levin	The Fundamentals of Bone Disease		
209—Wohl Aegerter	Unusual Problems in Bone Diagnosis		
210—Jacobson	The Vaterian Segment: Normal and Abnormal		

*** At 1:30 P.M. a special course will be given at the 22 Club, which is located on the ground floor, one flight below the lobby level, by Dr. R. McWhirter of Edinburgh, Scotland on "Simple Mastectomy and Radiotherapy in the Treatment of Breast Cancer."**

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M.** **September 29, 1960**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
301—Nice	Roentgen Manifestations of Collagen Diseases in the Chest	**321—Martin**	Low Intensity Radium Needles in the Treatment of Cancer
302—Lester	Roentgen Diagnosis of Operable Cardiac Lesions	**322—Murphy**	Radiation Therapy of Skin Cancer
303—Marshak	The Small Intestine		
304—Campbell	Radiology of the Urinary Tract	**323—Rubin Casarett**	Clinical Radiopathology for the Practicing Radiologist
305—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses	**324—Lenz**	The Fight Against Cancer in the U.S.S.R.
306—Lussier	The Roentgen Manifestations of the Peripheral Nervous System Tumors	**325—Watson**	Advantages of Supervoltage Radiation in Treatment of Cancer of the Cervix, Uterus and Ovary
307—Miller	Roentgen Examination of the Stomach and Small Bowel		
308—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
309—Weens Gay	Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes		

CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **September 30, 1960**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
401 O'Brien	Cysts and Tumors of the Mandible	**421—Guttmann**	The Management and Results in the Treatment of Inoperable Carcinoma of the Lung with Two Million Volt or Cobalt 60 Therapy as Compared to the Results with Orthovoltage or One Million Volt Therapy
402—Traub	Myelography—Technique and Interpretation		
403—Marshak	The Small Intestine and Duodenum		
404—Campbell	Radiology of the Urinary Tract	**422—Moss**	Radiotherapy of Carcinoma of the Breast—One Response to a Therapeutic Dilemma
405—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses	**423—Rubin Casarett**	Clinical Radiopathology for the Practicing Radiologist
406—Eyler Ormond	The Acute Abdomen		
407—Taveras	Anatomy of the Subarachnoid Cisterns in Relation to the Diagnosis of Tumors of the Posterior Fossa	**424—del Regato**	Leukemias, Lymphomas, and Hodgkin's Disease
408—Nelson	Bronchography		
409—Finby	Male Cystourethrography		
410—Egan Dodd	Mammography		

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Radiation Therapy. By Walter T. Murphy, M.D., Director of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo, New York; Consultant in Radiation Therapy, U. S. Veterans Hospital, Buffalo, New York; Associate Clinical Professor of Radiology, University of Buffalo Medical School. Cloth. Price, $25.00. Pp. 1041, with numerous illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia, Pa., 1959.

This large, well-written volume presents a systematic review of the anatomic, clinical-pathologic, therapeutic and prognostic aspects of the neoplastic entities of concern to the radiotherapist.

Introduced by an informative chapter on physical and biologic factors affecting radiation therapy, each subsequent chapter deals with an individual neoplastic entity or an appropriate anatomic region. Methods of treatment are discussed in considerable detail. Current techniques, as employed by the author and as reported by other workers, are fully described. Of considerable interest are the numerous, excellent clinical photographs depicting lesions before and often years after irradiation. The treatment factors of this older case material are presented in complete detail in the captions and provide a valuable correlation with late radiation changes. Critical comments by the author indicating desirable modifications of technique are included.

The numerous references appended to each chapter indicate the source material in the literature and represent a valuable feature. This is a book which everyone interested in the radiotherapy of cancer will wish to have.

Isadore Lampe, M.D.

An Atlas of Normal Radiographic Anatomy. Second edition. By Isadore Meschan, M.A., M.D., Professor and Director, Department of Radiology, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina; Consultant, Walter Reed Army Medical Center, Washington, D. C.; Formerly, Professor and Head of the Department of Radiology, University of Arkansas School of Medicine; with the assistance of R. M. F. Farrer-Meschan, M.B., B.S., M.D. (Melbourne, Australia), Research Associate, Department of Radiology, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina. Cloth. Price, $16.00. Pp. 759, with numerous illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia, Pa., 1959.

One cannot appreciate what is abnormal without first knowing what is normal. Based upon this general precept, the first edition of this book was published in 1951 and it has been revised and expanded in this, the second edition. What is presented is not, and was not meant to be, an encyclopedic work but a basic, practical and useful text correlating morbid anatomy and its roentgenographic appearance, the routine views needed to best study a given part and how they are obtained, and some of the more common variations of the normal which are encountered. A typical section includes a photograph of the morbid specimen, taken from a standard text book of anatomy, drawings of the necessary positioning and roentgenographic technique to adequately study the part in question, the resultant roentgenogram, and an accompanying labelled line drawing identifying what can be demonstrated on the roentgenogram. Charts, graphs and outlines are included covering ranges of the normal in development and roentgenographic appearance, mensurations for various relationships of anatomic structures, and criteria for diagnosis of some conditions.

Major areas of revision of or addition to the first edition include the addition of a chapter on radiation protection, the rewriting of the chapter dealing with bone growth and development in the light of more dynamic concepts, the inclusion of additional pneumoencephalographic, arteriographic and venographic information for the study of the brain, further material on the spine with greater emphasis on cervical myelography, advances in cardiovascular roentgenographic anatomy and phys-

iology, new concepts of the swallowing function and the esophagogastric junction, measurements in the relationships of the stomach to adjacent structures, some revision in biliary tract roentgenographic anatomy, and new techniques for examination of the colon. A chapter on the nature and production of roentgen rays and roentgenograms is included. Illustrations have been replaced and LogEtronic prints utilized wherever necessary.

The book very adequately fulfills its objective as a basic, general text on normal roentgenographic anatomy. It is to be noted that detailed information on special views and procedures is necessarily lacking and the bibliography included for possible further perusal is limited. Uncommon anatomic variations and the fringes of normal also are not covered and one should refer to texts devoted especially to these subjects.

This book should be included in the armamentarium of every radiology resident who wishes to be well informed in this specialized, but still general, field of medicine and should be referred to by the roentgen-ray technician desiring insight into his work. It also will be a useful tool to the anatomist, medical student and practitioner striving for a more dynamic concept of the anatomic relationships of the body's structures.

DONALD M. MONSON, M.D.

BOOKS RECEIVED

ATLAS OF ELECTROMYOGRAPHY. By N. Rosselle, M.D., Head of the Department of Electromyography, University of Louvain. Paper. Price, $5.00. Pp. 159, with 183 illustrations. Edouard Nauwelaerts Editions, 2, Place Cardinal Mercier, Louvain, Belgium, 1959.

TUMORS OF CHILDHOOD; A CLINICAL TREATISE. By Harold W. Dargeon, M.D., Attending Pediatrician and Chief, Pediatric Service, Memorial Hospital for Cancer and Allied Diseases; Attending Pediatrician, St. Luke's Hospital; Associate Professor of Clinical Pediatrics, Cornell University Medical College, New York. Cloth. Price, $20.00. Pp. 476, with numerous illustrations. Paul B. Hoeber, Inc., 49 East 33rd St., New York 16, N. Y., 1960.

MEDICAL PHYSICS. Volume III. Edited by Otto Glasser, Ph.D., Diplomate in Radiological Physics, American Board of Radiology; Professor of Biophysics, Frank E. Bunts Educational Institute; Head, Department of Biophysics, Cleveland Clinic Foundation; Member, Council on Medical Physics, American Medical Association. Cloth. Price, $25.00. Pp. 754, with numerous illustrations. Year Book Publishers, Inc., 200 East Illinois St., Chicago 11, Ill., 1960.

THE OFFICE ASSISTANT IN MEDICAL PRACTICE. Second edition. By Portia M. Frederick, Instructor, Medical Office Assisting, Long Beach City College; and Carol Towner, Director of Special Services, Communications Division, American Medical Association. Cloth. Pp. 407. W. B. Saunders Co., 218 West Washington Square, Philadelphia, Pa., 1960.

EDEMA; MECHANISMS AND MANAGEMENT; A HAHNEMANN SYMPOSIUM ON SALT AND WATER RETENTION. Edited by John H. Moyer, M.D., Professor and Chairman of the Department of Medicine, Hahnemann Medical College and Hospital; and Morton Fuchs, M.D., Assistant Professor of Medicine, Hahnemann Medical College and Hospital. Cloth. Pp. 833. W. B. Saunders Co., 218 West Washington Square, Philadelphia, Pa., 1960.

FUNDAMENTALS OF CLINICAL HEMATOLOGY. By Byrd S. Leavell, M.D., Professor Internal Medicine and Physician-in-Charge, Hematology Section, School of Medicine, University of Virginia; Attending Physician, University of Virginia Hospital; and Oscar A. Thorup, Jr., M.D., Associate Professor of Internal Medicine, School of Medicine, University of Virginia; Attending Physician, University of Virginia Hospital. Cloth. Pp. 503, with 71 illustrations. W. B. Saunders Co., 218 West Washington Square, Philadelphia, Pa., 1960.

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STUDIES IN RADIOTHERAPEUTICS. By J. S. Mitchell, C.B.E., M.D., F.R.S., Regius Professor of Physic, University of Cambridge. Cloth. Price, $9.00. Pp. 274. Harvard University Press, Cambridge 38, Mass., 1960.

RADIOLOGY AS A DIAGNOSTIC AID IN CLINICAL SURGERY. By Howard Middlemiss, M.D., F.F.R., D.M.R.D., Director of Radiology, United Bristol Hospitals; Lecturer-in-Charge, Department of Radiodiagnosis, University of Bristol. Cloth. Price, $7.50. Pp. 151, with 86 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Ave., Springfield, Ill., 1960.

LEUKAEMIA; RESEARCH AND CLINICAL PRACTICE. By F. G. J. Hayhoe, M.A., M.D. (Cantab.), M.R.C.P. (Lond.), Lecturer in Medicine, University of Cambridge; Honorary Physician, The United Cambridge Hospitals. Cloth. Price, $16.00. Pp. 335, with 12 colored plates and 196 black and white illustrations. Little, Brown & Co., 34 Beacon St., Boston 6, Mass., 1960.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Martinez, N., Léger, Jean-Louis, Ouimet, Denise, and Bertrand, Claude. L'opacification par voie carotidienne de l'artère cérébrale postérieure. (Opacification of the posterior cerebral artery via the carotid.) *J. Canad. A. Radiologists*, Dec., 1959, *10*, 51–55. (From: Hôpital Notre-Dame, Montréal, Québec, Canada.)

The authors stress the importance of knowledge of the anatomy of the cerebral vessels, particularly of the posterior cerebral artery, inasmuch as compression, displacement or deviation of this artery is indicative of serious disease, especially of tumor formation in that area. By the same token, they stress the importance of visualization of the posterior cerebral artery.

The authors' experience is based on 332 carotid arteriographies, performed between January, 1956 and August, 1958, on patients between the ages of three and sixty. Of these, 150 injections were made into the common carotid, 12 injections into the external carotid and 170 injections into the internal carotid.

Based on their early experience in studies of thrombosis of the carotid artery, the authors routinely include the entire neck on the roentgenogram. In this manner, they are able to determine accurately the site of injection and the position of the needle. Of 170 cases of internal carotid arteriography, visualization of the posterior communicating, as well as of the posterior cerebral, arteries occurred in almost 80 per cent of the cases.

Using pentothal anesthesia and a stellar block, a No. 17 needle is introduced at the level of C5, directed upwards approximately 2 cm. into the artery to the level of the fourth cervical vertebra. This ordinarily places the tip of the needle in the internal carotid or, at most, direct at the bifurcation, with the stream of the contrast medium flowing mainly into the internal carotid.

The authors believe, contrary to the ideas maintained by others, that a stellar block eliminates vasospasm and hypotension and that visualization of the internal carotid and, subsequently, of the posterior cerebral artery, is a direct result of their improved technique. This results in visualization of the posterior cerebral artery in 4 out of 5 cases.

The indications for this technique are all cases where there is a suspicion of extensive lesions of the temporal lobe, vascular malformations and tumors of the cerebellopontine angle or pathology of the occipital lobe.—*William H. Shehadi, M.D.*

Jost, Franz. Zur Klinik der basilären Impression. (Clinical features of basilar impression.) *Wien. klin. Wchnschr.*, April 4, 1958, *70*, 237–243. (From: Neurologisch-Psychiatrische Klinik der Universität, Innsbruck, Austria.)

The designation, basilar impression, refers to a skeletal anomaly of the base of the skull and the occipitocervical junction in which the region of the foramen magnum protrudes into the cranial cavity. In severe deformities the term, basilar invagination, is used.

The terms, platybasia and basilar impression, are often employed as synonyms. Some authors consider basilar impression and platybasia to be separate entities, the latter being characterized by flattening of the base of the skull with increase in the basal angle. Platybasia can occur without basilar impression and is often a relatively harmless anomaly, rarely associated with neurologic symptoms.

Basilar impression can be primary congenital and is either isolated or, more frequently, associated with other congenital abnormalities of the craniocervical region (assimilation of atlas, os odontoideum, Klippel-Feil syndrome, Arnold-Chiari syndrome and others).

Secondary basilar impression is encountered mainly in rickets, Paget's disease, osteogenesis imperfecta, etc., and is more common than the congenital variety. Eight case histories are presented in detail. In 4 cases, basilar impression of the congenital type was present, associated with neurologic signs resulting from compression of the cerebellum, brain stem and cranial nerves. The other cases had various disorders of the central nervous system; basilar impression was an accidental roentgenographic finding and was not responsible for the symptoms.

Roentgenologic examination demonstrates the following: (1) protrusion of the occipital condyles into the posterior fossa, visible on roentgenograms taken in the anteroposterior projection with angulation of the tube 35 degrees caudad (Towne's projection); (2) decrease of the basilar angle (angle between the planum sphenoidale and the clivus) which usually measures 120–150 degrees; this angle, according to Lindgren is of limited significance as it only shows the deformity at the midline, with little change in the slight basilar impression.

The following lines, drawn on lateral skull roentgenograms or frontal laminagrams, are helpful in showing the normal and pathologic relationships of the upper cervical vertebrae and the occiput: (1) Chamberlain's line, which joins the lower posterior edge of the maxilla with the posterior edge of the foramen occipitale magnum. The tip of the odontoid normally lies caudad to this line; (2) MacGregor's line, which joins the lower posterior edge of the maxilla with the most caudal point of the occiput. The tip of the odontoid reaches a few millimeters above this line; and (3) Fischgold's lines, which are drawn on anteroposterior laminagrams of the skull. The bimastoid line connects the lower ends of both

mastoid processes and passes normally through the atlanto-occipital joints. The biventer line is drawn through the joints of attachment of the digastric muscles and lies 1 cm. above the tip of the odontoid process.—*Karol E. Matzinger, M.D.*

Valentino, Vincenzo. Combined intraventricular and intracerebral hydatid cysts; report of a case. *Radiology*, Aug., 1959, *73*, 250–252. (Address: Via Morgantini 3, Naples, Italy.)

An unusual case of multiple intracerebral and intraventricular hydatid cysts occurring in a patient known also to have hydatid disease of the lung is presented.

The author stresses the importance of the preoperative diagnosis of these intracranial hydatid cysts, since a surgical approach to the lesion without a knowledge of its true nature could lead to rupture of the cyst and to wide dispersion of hydatids. Although angiography is considered by some to be the most reliable and safest method of establishing the diagnosis of avascular space-occupying lesions of cystic type, the author believes that the judicious use of ventriculography may be quite helpful for the diagnosis of small cysts and for those cases in which angiography is known to be of little value, *i.e.*, intraventricular cysts.—*John S. Alexander, M.D.*

Neck and Chest

Philp, T. Some congenital cysts and fistulae of the neck. *J. Fac. Radiologists*, Oct., 1959, *10*, 186–196. (From: Radiodiagnostic Department, Royal Infirmary, Edinburgh, Scotland.)

Congenital cysts and fistulae occurring in the neck may be classified under (1) those appearing in the midline and (2) those arising in the lateral aspect of the neck.

In the midline group, one finds the thyroglossal cyst and the thyroglossal fistula. These lesions are the result of incomplete obliteration of the thyroglossal duct during the development of the thyroid gland. Residual cell-rests may give rise to aberrant thyroid glands, which will be located in the line of the duct, and residual segments of unobliterated duct may give rise to thyroglossal cysts. Fistulae may develop following the rupture of such cysts, but a long sinus tract may be due to a persisting thyroglossal duct. This may communicate with the pharynx at the foramen caecum, but any external orifice in the neck will be secondary, that is, due to infection or surgical intervention. Hence, it is possible to have a complete fistula, a sinus with an internal opening, a sinus with an external opening, or a cyst. The most common site for a cyst is immediately below the hyoid bone. This usually appears during childhood or early adult life, varying in size from a pea to a walnut. Treatment of both cysts and fistulae is by complete excision. Signograms provide a ready means for determining the extent of the lesion.

In the lateral group the most common condition is the branchial or lateral cervical cyst, sinus or fistula. As in the case of the thyroglossal lesions, the branchial fistulae may be complete or incomplete, with either internal or external opening, or there may be a cyst without external communication. The external skin orifice is always found along the anterior border of the sternomastoid muscle, just above the suprasternal notch. The internal orifice is in the region of the tonsillar fossa. Clinically, the fistula is manifest by the external orifice in the typical situations; the tract may be felt as a cord-like structure, and the sinus may discharge a watery secretion. The branchial cyst forms a smooth, tense, fluctuant swelling 1 to 10 cm. in diameter, deep to the anterior border of the sternomastoid below the angle of the mandible. The cyst slowly enlarges and usually presents by adolescence or early adult life. The contents of these cysts may be aspirated and replaced by a contrast medium for roentgenographic visualization. Branchial fistulae also may be outlined with contrast material and their full extent determined. Treatment is by complete excision.

The only other condition of importance in this series is the cystic lymphangioma, which, though not a common condition, is found in the root of the neck and occasionally in the axilla and groin. In the case of the neck lesion, not infrequently there is found extension into the superior mediastinum, producing certain characteristic roentgenographic appearances. The term "cystic hygroma" has been applied to these lesions. The multilocular cystic lymphangiomata do not normally present as a roentgenologic problem unless they extend into the chest. They become obvious during the first year (80 per cent), forming large cystic masses extending to the surface. The roentgenographic demonstration of these lesions is confined to plain roentgenograms of the neck and chest to determine the degree of mediastinal extension. The solitary lymphatic cyst is found in the supraclavicular region lateral to the sternomastoid, forming a fluctuant, deeply situated, soft tissue swelling, often occurring during adolescence or early adult life, perhaps because of secondary infection. Following aspiration, the cyst may be filled with contrast material for roentgenographic demonstration.—*Samuel G. Henderson, M.D.*

Williams, Howard, and O'Reilly, R. N. Bronchiectasis in children: its multiple clinical and pathological aspects. *Arch. Dis. Childhood*, June, 1959, *34*, 193–201. (From: Clinical Research Unit, Royal Children's Hospital, Melbourne, Australia.)

The authors discuss the controversy concerning the etiology, pathology and pathogenesis of bron-

chiectasis. The two principal and most widely accepted theories of pathogenesis are: (1) the disease is primarily due to bronchial damage from infection, the weakened bronchi then dilating; and (2) pulmonary collapse is the primary cause, bronchial dilatation initially being due mainly to secondary mechanical factors. Pathologic studies have revealed a wide variety of lesions. In some the essential structure of the bronchi and bronchioles was completely destroyed by infection; in others the component parts of the bronchial wall were normal. Collapse of the affected lobes or segments was a feature in some, but not in others.

Two explanations are possible: (1) there are all grades of severity of a disease entity, which may be caused by a variety of infections; and (2) there are a number of separate diseases, each with a different pathology and clinical pattern, but all with the one common gross morphologic feature of bronchial dilatation.

The authors report a series of 650 patients with bronchiectatic lesions. They have studied 241 of them from the inception of the disease process and have followed them for periods of four to ten years. The following classification is submitted:

1. Subacute pyogenic pulmonary collapse	37
2. Nonspecific infective bronchiolitis and/or interstitial pneumonia	57
3. Fibrocystic disease of the pancreas	56
4. Primary pulmonary tuberculosis	53
5. Congenital malformations of the bronchial tree	16
6. Miscellaneous lung infections	22
Total	241

The first two entities are described in some detail.

(1) *Subacute pyogenic pulmonary collapse* has been arbitrarily defined as a pyogenically infected collapsed lobe or segment which has been collapsed for a period between six weeks and one year. In 37 children in this group, nonspecific respiratory infection was the cause of collapse in 16, pertussis in 11, measles in 2, allergic bronchitis in 4, a foreign body in 2 and postoperative causes in 2.

The two outstanding clinical features were persistent cough following the initial respiratory infection and constitutional symptoms of ill health. Roentgenologic signs of lobar collapse were unequivocally demonstrated in all patients, the lesion being unilateral in 27 and bilateral in 10.

Radiopaque oil instilled into the bronchial tree demonstrated dilated crowded bronchi. The dilatation affected the middle divisions of the bronchial tree, and was saccular in 7 instances and cylindrical in 30.

Bronchoscopic examinations revealed mucopus in the affected lobar bronchus but the bronchial lumen was not occluded by a mucus plug, mucosal swelling or glandular pressure.

Resolution of the process was complete in 16 patients, while 7 were left with permanent collapse and bronchiectasis. Partial resolution occurred in 14 patients, leaving varying degrees of patchy bronchiectasis and collapse. Seven patients with persistent cough and sputum were cured by lobectomy. Four became symptom free one to two years after the initial infection and have dry bronchiectasis.

(2) *Nonspecific infective bronchiolitis and/or interstitial pneumonia* was observed in 57 patients with bronchiectasis. Pulmonary collapse is not an essential pathologic feature, although it may occur.

The disease is seen primarily in the age group from one to three years. Persistent cough, continuous or intermittent nasal discharge, sudden attacks of fever, and general ill health were the dominant features in all patients.

The roentgenographic findings varied moderately with either a prominent bronchovascular pattern or a mottled diffused bronchopneumonia. Lobar or segmental collapse was present in only 10 patients, but developed in 9 others at a later date.

The severity of the illness appeared to parallel the pattern of bronchiectatic changes. The most severely affected had generalized bronchiectasis which was fusiform, cylindrical or saccular in type. A group in whom the symptoms were much less severe had bronchiectasis which was either localized or patchy in distribution. Changes were observed to develop in a group of 27 patients in a lobe or segment previously demonstrated as normal bronchographically. Once the bronchiectatic lesions were present, they remained and reversible changes were never observed.

Treatment by chemotherapy, postural coughing and physiotherapy resulted in complete or partial resolution of the collapse and bronchiectasis in many patients with subacute collapse. Similar treatment was much less effective in the other patients owing to their inability to carry out effective postural coughing and physiotherapy, as the majority were under three years of age. In addition, chemotherapy could not be used specifically as pathogenic organisms were not isolated in most patients.—*James F. Martin, M.D.*

Müller, W., and Musshoff, K. Ampulläre Bronchiektasen, eine seltene Missbildung der Bronchien. (Ampullary bronchiectases, a rare malformation of the bronchi.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1959, *91*, 701–708. (Address: W. Müller, Mediz. Univ.-Klinik, Röntgen-Rad.-Abteilung, Freiburg im Breisgau, Germany.)

As early as 1880, the pathologist Grawitz found bronchiectasis in newborn infants at autopsy. Nevertheless, the question of congenital bronchiectasis in the adult has not yet been clarified. The authors re-

port 3 adult cases with ampullary bronchiectasis which they believe to be of congenital origin.

The first patient was a female, aged thirty-four, with a history of frequently recurring attacks of pneumonia from the age of six months, and with arthritic manifestations of the fingers for the preceding ten years. Chest roentgenograms showed increased pulmonary markings in the middle and lower lung fields. Bronchography revealed ampullary bronchiectasis extending to the peripheral subsegmental bronchi bilaterally, but predominantly in the larger bronchi. There was also cylindric bronchiectasis in both lower lobes.

The second patient was a janitor, aged forty-one, with a history of pneumonia at the age of twenty. In recent years he had noticed a cough with increasing frequency, expectoration on arising and loss of appetite and weight. The chest roentgenogram showed ring formations in the right upper lobe and apex. Bronchography revealed ampullary dilatations in the left lower lobe subsegmental bronchi of the first, second and third order, but not in the periphery. In the right upper lobe, there was cystic dilatation of the bronchi and chronic deforming bronchitis. The diagnosis was a combination of ampullary bronchiectasis and honeycomb lung (Wabenlunge).

The third patient was a housewife, aged forty-four, with a history of recurrent attacks of pneumonia over a period of several years. The chest roentgenogram showed cystic radiolucencies in areas of bronchopneumonic infiltrates, especially throughout the left lung, but also in the right upper lobe. Bronchography revealed multiple cysts throughout the left lung and in the right upper lobe, also cylindric and saccular bronchiectasis in the left lower lobe and ampullary changes in the right upper lobe.

Acquired bronchiectasis usually is of the cylindric or saccular type. It is located in the periphery of the lungs and results in the bronchographic appearance of a "leaveless tree" with absence of alveolar filling.

In the congenital type, however, the periphery is clear. The ampullary and varicose changes are located in the larger, more proximal, bronchial segments. The condition is relatively rare, having been observed in only 3 of 65 cases of bronchiectasis in this series. However, it may not be as rare as has been assumed, since most cases have no superimposed peripheral involvement and, therefore, remain asymptomatic.

The ampullary formations tend to retain secretions, which causes delay of bronchial drainage. Therefore, development of secondary bronchiectasis is not surprising, especially in the basal segments where postural drainage is impaired.

Form and localization of the ampullae suggest a congenital nature with a primary weakening of the bronchial walls. Kartagener proves the congenital origin by associated malformations elsewhere, and by a combination of sinusitis and cystic lung changes.—*Ernest Kraft, M.D.*

Fraser, Robert G., and Wortzman, George. Acute pneumococcal lobar pneumonia: the significance of non-segmental distribution. *J. Canad. A. Radiologists*, Sept., 1959, *10*, 37–46. (From: Royal Victoria Hospital, Montreal, Quebec, Canada.)

There has been a tendency for radiologists to relate all localized pulmonary diseases to the bronchial supply of the involved area, thus inferring that the abnormality is segmental. The authors point out that this practice may be subject to error and can give rise to incorrect roentgenologic interpretation. Many sublobar opacities produced by acute pneumococcal pneumonias are nonsegmental and should not be ascribed to a specific bronchial distribution. This nonsegmental anatomic distribution is not necessarily due to an atypical bronchial supply.

It has been demonstrated experimentally that acute exudative pneumonias spread circumferentially by direct dissemination of bacteria-laden edema fluid from alveolus to alveolus through the intra-alveolar pores, across lobular and intralobular septa, and by gravity effects through the bronchioles.

The authors report that 44 of 56 cases of acute pneumococcal pneumonia showed nonsegmental sublobar consolidation. These were frequently peripheral, generally round in contour except where the margin abutts against a fissure, homogeneous in density, and with a distribution which reflected the influence of gravity on the intrabronchial aspirate. Peripheral spread across segmental boundaries within the lobe was seen to occur.

Recognition of this characteristic pattern of sublobar consolidation facilitates differential diagnosis by distinguishing the process of alveolar exudation from those segmental densities due to obstructive pneumonitis of bronchial origin (atypical pneumonias), bronchial cancer, and the peribronchial and interstitial inflammatory conditions which produce segmental patterns (streptococcal and staphylococcal bronchopneumonias).—*John A. Campbell, M.D.*

Small, Maurice J. Late progression of pulmonary coccidioidomycosis. *A.M.A. Arch. Int. Med.*, Nov., 1959, *104*, 730–740. (Address: Veterans Administration Hospital, East Orange, N. J.)

Coccidioides immitis produces a primary benign type of pulmonary infection and rarely a serious disseminated form. The primary cavity is usually associated with surrounding pneumonitis and usually closes spontaneously. The residual pulmonary cavity appears as a thin walled structure without surrounding tissue reaction and this type of cavity and the coccidioma have not been considered dangerous to the patient, although both may contain fungi and the former may frequently be the source of hemoptysis. Although there have been records of thousands of

military patients with coccidioidosis, there has been no report of dissemination occurring in a patient with cavitation, according to authorities writing on this subject in 1948.

Four cases of pulmonary coccidioidomycosis are reported in detail and these cases seem to indicate that: (1) the disease may not manifest itself until a long time after exposure; (2) it may undergo late, continued, slow progression over a period of years; and (3) it may spread by the bronchial route, from cavities previously considered benign, to produce serious disease in later years. Since the cases reported were in persons residing in the eastern part of the United States, it is obvious that this disease must be considered in all cases of obscure pulmonary disease from any section of the country.—*Richard E. Kinzer, M.D.*

Grosse-Brockhoff, F. Die Lungenzirrhose. (Pulmonary cirrhosis.) *Deutsche med. Wchnschr.*, April 18, 1958, *83*, 677–681. (Address: I. Med. Klinik d. Med. Akademie, Moorenstr. 5, Düsseldorf, Germany.)

The term, pulmonary cirrhosis, is used here for the disease entity known on the North American continent as the Hamman-Rich syndrome, a diffuse, progressive, interstitial pulmonary fibrosis. The condition was first described by Rindfleisch in 1897 under the name of pulmonary cirrhosis. Von Hansemann, in 1898 and 1915, described it as a separate disease entity under the term, lymphangitis reticularis. It was rediscovered by Hamman and Rich in 1935 when they described 3 cases, to which they added a fourth in 1944.

The disease can occur in an acute form with a rapidly fatal course, but in the majority of cases the course is chronic and progressive over a period of several years. The duration in the cases reported was from one month to over twenty years; as the onset is usually insidious, it is impossible to determine its exact duration. There is no predilection for either sex. Approximately 40 cases verified by autopsy have been published to date. Four additional verified cases are reported by the author.

The disease represents a well defined pathologic entity, with a certain resemblance to the pulmonary manifestations of the collagen diseases. The symptoms of dry cough, gradually increasing dyspnea and cyanosis are stressed. A cor pulmonale, with electrocardiographic signs of right heart hypertrophy and right heart failure, develops. The outcome is always fatal, with death due to respiratory and right heart failure.

The roentgenographic findings consist of diffuse reticular or strand-like markings or patches of increased density. The appearance can change in the course of the disease. The process is usually symmetric and shows a predilection for the midlung fields, sometimes having a butterfly-like effect.

The sedimentation rate can be normal or increased. Slight to moderate polycythemia is frequent as well as an increase in gamma globulins.—*Karol E. Matzinger, M.D.*

Sprunt, William H., Peters, Richard M., and Holder, David L. The significance of alterations in the lung arterial pattern. *Radiology*, July, 1959, *73*, 1–8. (Address: W. H. Sprunt, School of Medicine, University of North Carolina, Chapel Hill, N. C.)

Roentgenographic procedures which will aid in the evaluation of pulmonary abnormalities and in the selection of patients for surgical treatment are described. To demonstrate the value of information obtained in this manner, venous and aortic angiography was performed in dogs after the blood supply had been altered in three ways: (1) by transection and suture of a main bronchus, (2) by ligation of a main pulmonary artery, and (3) by anastomosis of the left subclavian artery to the pulmonary artery.

In animals in which the bronchus had been transected, there was collapse of the lung, with a shift of the mediastinum toward that side. Aortograms revealed no filling of the bronchial arteries on either side. Venous angiography provided good visualization of the pulmonary arteries on the unoperated side of the chest. These vessels were two or three times larger than normal and were widely separated. The pulmonary arteries on the opposite side were much smaller, less distinctly outlined and crowded together. Satisfactory anastomosis of the transected bronchus was associated with re-expansion of the collapsed lung and return of the pulmonary arteries to normal size and position.

Ligation of the main pulmonary artery did not result in collapse of the lung or mediastinal shift. Venous angiography demonstrated the effectiveness of the ligation. There was slight increase in the size of the vessels on the normal side. Retrograde aortograms showed good filling of the intercostal arteries bilaterally; they were a little more distinct on the side of the ligated vessel than on the normal side. A network of dilated bronchial arteries was present on the side of the ligated pulmonary artery.

Venous angiography, after a left subclavian pulmonary artery anastomosis had been performed, produced good filling of the right pulmonary artery. Retrograde aortography satisfactorily demonstrated the systemic pulmonary artery shunt and the left pulmonary artery. The intercostal arteries were easily identified but no bronchial arteries could be noted.

These methods and findings can be applied to the evaluation of pulmonary disease in clinical practice. In patients with pulmonary stenosis, bronchiectasis, carcinoma, tuberculosis, and emphysema, there is enlargement of the bronchial arteries and their anastomoses with the pulmonary arteries. No in-

crease in the bronchial arterial collateral circulation is demonstrable by angiography in uncomplicated chronic atelectasis. Aortography is a means of evaluating the physiologic status of collapsed lung due to traumatic rupture of a bronchus. Demonstrable bronchial arteries, in the presence of abnormal arterial oxygen saturation, are an indication that the lung is diseased and that repair of the bronchus would be contraindicated. Lack of visualization of the bronchial arteries and near normal oxygen saturation suggest that the lung is essentially normal and that repair of the bronchus would probably result in a functioning lung. In patients with extensive chronic pulmonary disease but with incomplete collapse of one lung, the question of operability can be resolved best by pulmonary arteriographic studies. By this procedure the vascular status of both lungs can be determined, and surgery can be recommended if the contralateral lung is functioning well enough to permit resection of the abnormal lung.—*A. W. Sommer, M.D.*

Gamalero, P. C., Bono, G., and Grassi, E. Observations bronchographiques des alterations de l'arbre tracheo-bronchique dans les angio-cardiopathies congénitales. (Bronchographic observations of the changes of the tracheobronchial tree in congenital angiocardiopathies.) *J. belge de radiol.*, 1959, *42*, 551–568. (From: Clinique de Pédiatrie de l'Université de Turin and Hôpital des Enfants "Regina Margherita," Institut de Radiologie Médicale, Turin, Italy.)

In view of the increased incidence of studies of congenital angiocardiopathies, the problem of accompanying respiratory distress, concomitant with cardiovascular malformations, becomes all the more important.

An abnormal, or heterotopic vessel, may compress the trachea and/or esophagus, causing partial obstruction of these organs. In this connection, the malformations of interest are double aortic arch, right sided aorta with left sided ligamentum arteriosum, and aberrant subclavicular vessels. The chances of survival of these subjects are limited, because of the respiratory distress. Other congenital anomalies and malformations of significance in this type of disturbance may occur involving the bronchopulmonary system.

The studies in this paper are based on bronchographic examinations permitting tracheobronchial visualization all the way to the extreme periphery. This permits satisfactory and detailed evaluation of the tracheobronchial tree and its relationship to the adjacent cardiovascular structures.

The authors base their studies on a series of 40 children between the ages of six and twelve with diverse congenital angiocardiopathic conditions. These are classified as tetralogy of Fallot, 11 cases; tetralogy of Fallot (Corvisart variety), 1 case; truncus arteriosus communis, 1 case; interauricular communication, 10 cases; interventricular communication, 11 cases; and persistence of the arterial canals, 6 cases. The diagnoses in these patients were confirmed by catheterization and angiocardiography.

The evening prior to the bronchographic examination a sedative was administered orally (nembutal or elixir of luminal) and repeated the following morning. Local anesthesia of the oral pharynx, the larynx and the trachea as well as the bronchi was obtained with formocaine 0.5 per cent and later by instillation into the pharynx of 0.25 per cent formocaine spray. Under fluoroscopic control, the contrast medium (dionosil and propyliodon) was injected. No adverse reactions were observed with this technique.

The findings may be divided into three major groups: (1) anatomic or morphologic alterations, (2) anatomico-functional or physiologic changes, and (3) congenital tracheobronchial anomalies associated with cardiovascular malformations. Involvement of the trachea and bronchi consists of: (1) deviation of the trachea and major bronchi, (2) compression of the trachea and major bronchi, and (3) a combination of these. Other findings are bronchial rigidity, compression, displacement, bronchiectasia, bronchial divisions and bifurcation, both lobar and segmental. These can be explained on the basis of compression due to the congenital anomaly, with enlargement and variations in the shape of segments of the heart. Lesions involving the peripheral bronchi are noted mostly in anomalies associated with increased flow of pulmonary circulation, such as occurs in interauricular and interventricular septal defects, and persistence of Botallo's duct.—*William H. Shehadi, M.D.*

Davis, Lawrence A., and Vermillion, Margaret. The relationship of the cardiac silhouette to altered respiratory dynamics in congenital heart disease in infants. *Radiology*, July, 1959, *73*, 49–55. (Address: L. A. Davis, 226 E. Chestnut St., Louisville 2, Ky.)

Infants with congenital heart disease will frequently show a hyperinflation of the lungs on chest roentgenograms. This pulmonary hyperventilation is thought to be compensatory in nature and due to an attempt of the body to overcome the poor oxygenation of the blood. Since inspiration is an active process and expiration a passive one, a small residual increment of air is trapped in the lungs during each respiratory cycle when the respiratory rate is so high that there is not time for full expiration. A similar explanation applies, in part, to those patients with pure inflammatory disease of the lungs, where the hyperventilation is a compensatory mechanism as well as secondary to increased airway resistance.

It should be remembered that the actual outline

and configuration of the heart and its relationship to the chest wall can be seriously disturbed merely because of change in the thoracic contours and not necessarily as a result of any intrinsic cardiac abnormality. Because of this, it is evident that in analyzing the cardiac contour in infants the state of pulmonary inflation at the time of the exposures must be considered.

These changes incident to overaeration may increase as the disease advances or may subside secondary to either surgical correction or medical therapy. —*Donald N. Dysart, M.D.*

Eyler, William R., Wayne, David L., and Rhodenbaugh, John E. The importance of the lateral view in the evaluation of left ventricular enlargement in rheumatic heart disease. *Radiology*, July, 1959, *73*, 56–61. (Address: W. R. Eyler, The Henry Ford Hospital, Detroit 2, Mich.)

Surgical relief for patients with mitral valve lesions resulting from rheumatic fever is more effective in mitral stenosis than in mitral insufficiency. Consequently, the estimation of the degree of insufficiency in a mitral lesion is an important factor in predicting benefit from surgery. The authors have found that the most reliable single indicator of mitral insufficiency is the detection of left ventricular enlargement in the lateral view of the chest.

The roentgenograms of 214 cases of rheumatic valvular disease, surgically explored, were examined. In mitral stenosis, the lateral roentgenogram usually shows only a short segment of heart in contact with the left leaf of the diaphragm and the posterior aspect of the heart presents a sigmoid shape. There is often a clear space between the heart and the barium filled esophagus and the heart does not protrude more than 10 mm. behind the upper end of the inferior vena cava. In mitral insufficiency, the posterior enlargement of the left ventricle causes the segment of heart in contact with the diaphragm to lengthen. The posterior aspect of the heart now produces a convex shadow posteriorly and this usually extends at least 15 mm. beyond the upper end of the inferior vena cava.

A cardiothoracic ratio (C/D ratio) was used in this study to estimate left ventricular size and was obtained from the lateral chest roentgenogram. The internal diameter of the chest (D) represents the distance between the anterior and posterior pleural surfaces. The cardiac segment (C) is that distance from the anterior pleura to the posterior point of contact between the heart and the left leaf of the diaphragm. Although definite values should not be rigidly employed, a C/D ratio of 0.42 or below usually indicates pure mitral stenosis without associated mitral insufficiency or aortic stenosis. In the presence of a huge left atrium, this method of measurement cannot be applied.

It is emphasized that the proper evaluation of cardiac chamber size requires the use of all four projections and fluoroscopy. When the roentgen information agrees with the clinical, further study such as left heart catheterization is not required.—*Edward B. Best, M.D.*

Herbst, M., Hartleb, O., and Bock, K. Klinische und angiokardiographische Bestätigung des Bernheim-Syndroms. (Clinical and angiocardiographic confirmation of the Bernheim syndrome.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1959, *91*, 679–689. (Address: M. Herbst, Chirurg. Klinik der Univ. Leipzig, Liebigstrasse 20 a, Leipzig, East Germany.)

In 1910, Bernheim postulated that an enlarged left ventricle could encroach upon the interventricular septum to such an extent as to cause right heart failure. In cases with left ventricular hypertrophy, the proper sequence of decompensation would be left heart failure with pulmonary congestion prior to right heart failure. In the Bernheim syndrome, however, the regular cycle is interrupted, pulmonary congestion is bypassed and right heart failure occurs at an early stage of the cardiodynamic disorder.

The Bernheim syndrome has been subjected to criticism in the past five decades and its validity has not been accepted by numerous authors. Neither the opponents nor the proponents, however, have been able to offer indisputable proof of their contentions. The syndrome has been corroborated by autopsy findings; but opponents have ascribed the crowding of the right ventricle to postmortem changes.

In recent years cardiac catheterization with pressure differential studies and angiocardiography has definitely established a sound basis for the theory of the Bernheim syndrome. Five cases are briefly reported and well illustrated in which pressure in the pulmonary artery was lower than in the right ventricle, the latter being crowded by a bulging septum. The cause of left ventricular hypertrophy was supravalvular aortic stenosis, aortic valve stenosis and aortic hypoplasia, respectively, in 3 cases and coarctation of the aorta in the remaining 2 cases.

The Bernheim syndrome is not rare if asymptomatic and borderline cases are included. It can be discovered in early life as seen in a four year old child of the present series. The authors recognize three stages: (1) crowding of the right ventricle, but only little pressure differential between the right ventricle and pulmonary artery, and absence of symptoms; (2) insufficiency of the right heart with marked narrowing of the right ventricle but without left heart failure; and (3) failure of the left ventricle.

Diagnosis of the Bernheim syndrome can be established on the basis of angiocardiographic findings and a pressure gradient between the right ventricle and the pulmonary artery which suggests a stenosis of

the right ventricular outflow tract. In the differential diagnosis, additional congenital heart lesions, especially septal defects with left to right shunt, have to be excluded.—*Ernest Kraft, M.D.*

Singleton, Edward B., McNamara, Dan G., Leachman, Robert D., Cooley, Denton A., and Chau, Paul M. Radiological evaluation of isolated ventricular septal defects before and after surgical closure. *Radiology*, July, 1959, *73*, 37–48. (Address: E. B. Singleton, Texas Children's Hospital, Houston 25, Texas.)

The authors report the results of a study of 144 patients with isolated ventricular septal defect studied at The Texas Children's Hospital. Operative closure of the defect was performed in 133 of these patients. Preoperative roentgenograms were evaluated in respect to cardiothoracic ratio, heart size, size of the left atrium, size of the pulmonary artery and prominence of the pulmonary vasculature. Postoperative studies with reference to these same factors were made immediately after operation, and at intervals up to two years following surgical closure.

The patients were placed in groups which were based arbitrarily on the ratio of the systolic pulmonary artery pressure to the systolic systemic pressure and the magnitude and direction of the shunt through the ventricular defect. All patients in whom the pulmonary systolic pressure was less than 50 per cent of the systemic pressure were designated as Group I; patients in whom the systolic pulmonary pressure was between 50 and 85 per cent of the systemic pressure constituted Group II. Both of these groups had a left-to-right shunt. In Group III there was a large left-to-right shunt; Group IV had an essentially balanced shunt; Group V had only a right-to-left shunt at the ventricular level. Pulmonary systolic pressure in Groups III, IV, and V was nearly equal to or even greater than the systemic systolic pressure.

With a normal pulmonary artery pressure and a small shunt, the roentgenographic appearance of the heart and pulmonary vascularity are usually normal. With elevation of the pulmonary artery pressure beyond 50 per cent of the systemic pressure and with a large left-to-right shunt, the left atrium is enlarged and the pulmonary artery and pulmonary vascularity become more prominent. The degree of the left atrial dilatation is a reliable index of the effective pulmonary blood flow. Left ventricular enlargement predominates, but a certain number of cases show combined ventricular hypertrophy.

Those cases in which the pulmonary systolic pressure is approximately equal to the systemic pressure, but with a large left-to-right shunt, show an accentuation of the features mentioned in the previous group, but there is a larger degree of right ventricular hypertrophy and dilatation. With equal pressures and a small left-to-right shunt or a moderate right-to-left shunt, the heart is smaller and the left atrium is less prominent, although right ventricular hypertrophy is greater. The pulmonary artery segment and the right and left branches are often unusually prominent.

In the group in which the shunt is entirely right-to-left, the over-all size of the heart is frequently normal or below normal; left atrial enlargement is unusual and pulmonary vascular markings are often decreased in prominence; the pulmonary artery segment remains large.

Postoperatively, the sequence of roentgenographic changes is essentially the same for all groups. Immediately following operation there is an increase in heart size. The greatest enlargement was found in those cases (Groups I and V) in which the preoperative cardiac-thoracic ratio was essentially normal. Between two and six months, and continuing up to a year, following surgery a regression in heart size had begun in all groups, with the greatest improvement occurring in those patients who preoperatively had the largest hearts. The pulmonary artery segment showed a prompt decrease in convexity. Three-fourths of all patients showed an increase in the pulmonary vascularity.—*A. W. Sommer, M.D.*

Mellins, Harry Z., Kottmeier, Peter, and Kiely, Brian. Radiologic signs of pericardial effusion; an experimental study. *Radiology*, July, 1959, *73*, 9–17. (Address: H. Z. Mellins, 450 Clarkson Ave., Brooklyn 3, N. Y.)

The authors present the results of an experimental study in which opaque pericardial effusions were produced in dogs by the injection of diodrast into the pericardial cavity. The animals were then examined in the recumbent, erect and four decubitus positions. Roentgen kymograms were obtained to record the nature of the pulsations. These studies showed that the pericardial fluid collected anteriorly, superiorly, laterally and, to a lesser degree, inferiorly, with almost no posterior localization. The changes in position of the animals were found to have little or no effect on the location of the fluid. The reason for this is due evidently to the fixation of the posterior heart surface to the soft tissues of the posterior mediastinum by the six blood vessels which enter the heart posteriorly.

The kymographic studies revealed that the pulsations of the anterior pericardial surface were damped while the anterior border of the heart just beneath it showed good pulsations. The barium filled esophagus posteriorly showed good transmitted cardiac pulsations. This differential damping of the cardiac pulsations is thought to be a dependable roentgenographic sign of pericardial effusion.

Two cases are discussed in which the procedures of fluoroscopy and kymography helped to make the diagnosis of pericardial effusion. It is believed by the authors that, as more experience accumulates, simple

fluoroscopy will suffice to make the diagnosis.—*Donald N. Dysart, M.D.*

Leger, L., Montète, P., and Guyet, P. Valeur et signification des varices oesophagiennes; voies anastomotiques fonctionnelles ou culs-de-sac? (Value and meaning of esophageal varices; functional anastomoses or cul-de-sacs?) *Presse méd.*, Dec. 25, 1959, *67*, 2272–2275. (From: Hôpital Lariboisière, Paris, France.)

Esophageal varices are classically considered as spontaneous portacaval anastomoses. The authors believe that while this may be true anatomically, they have minimal functional value, and are only a plug-region—a zone of portal stasis in which the tensional jerks of the portal system are broken. The following evidence is cited: (1) they can be excised without deleterious effects; (2) they fade out at higher levels without gross connection with the azygos vein or the mediastinal and periesophageal veins; (3) they may be distended by injection during surgery for a ruptured varix; (4) in splenoportography they are rarely filled, although a coronary reflux is shown; and (5) after a portacaval shunt, the coronary reflux is relieved almost immediately, whereas the esophageal varices disappear much more slowly.

The authors suggest that there may be two ascending systems: one from the coronary vein only, which is submucous and of little functional value; the other a periesophageal one supplied from the coronary and perisplenic vessels, constituting a valuable path for anastomosis with the azygos system.—*Frank A. Riebel, M.D.*

Zdansky, E. The anatomical and functional basis of the gastro-oesophageal reflux as seen radiologically. *German Med. Monthly*, Oct., 1959, *4*, 331–334. (Address: Bügerspital, Basle, Switzerland.)

Failure of the gastroesophageal closing mechanism with resultant reflux of gastric contents may lead to serious complications such as esophagitis, stenosis and shortening, peptic ulceration, and even the development of carcinoma of the esophagus. The effective sphincteric action at the gastroesophageal junction is believed to depend upon the correct interplay of a whole series of anatomic and functional factors which prevent the reflux of gastric contents into the esophagus.

Reflux occurs with such anatomic changes as enlargement of the esophageal diaphragmatic hiatus, insufficient fixation of the esophagus and gastric fundus by the phrenoesophageal ligament, loss of the normal acute angle between the cardiac antrum of the esophagus and gastric fundus, and upper subluxation of the stomach through the diaphragm into the chest.

Certain functional changes also enhance reflux of gastric contents into the esophagus. First, there is the pressure gradient in the epiphrenic ampulla as affected by respiration and diaphragmatic position with resultant sphincteric-like actions. There is also the elastic recoil of the contractile esophagus and the intra-abdominal pressure which, together with other mechanical weaknesses, favor the herniation of the gastric fundus and esophagus antrum with resultant reflux. Stimulation of various abdominal organs reflexly causes shortening of the esophagus via the vagus nerve, *i.e.*, gallbladder disease.

Organic shortening of the esophagus by relentless traction on the attachments of the esophagus to the hiatus and on the fundus may convert an intermittent subluxation into a fixed hernia and thus initiate a vicious circle favoring reflux. Similarly, reflux frequently causes shortening or a tube-like rigidity of the esophagus brought about by esophagitis, and these changes in turn cause further reflux.—*John A. Campbell, M.D.*

Abdomen

Rooney, Donald R. Aberrant pancreatic tissue in the stomach. *Radiology*, Aug., 1959, *73*, 241–244. (Address: Department of Radiology, Emory University Hospital, Atlanta 22, Ga.)

Aberrant pancreatic tissue in the stomach, a benign condition, is described. Seven additional cases with illustrations and brief descriptions are added to the 358 previously reported by various authors. The most common symptom is said to be epigastric pain, with pyloric obstruction next in order.

The author discusses the roentgenographic findings and emphasizes that the distinguishing feature is a regular rounded filling defect with a central dimple, which represents the central duct draining the aberrant tissue. These tumors are found most often in the antrum or pylorus, and the author urges that, when a 1 to 2 cm. filling defect is noted in this area, a very careful search be made for the central pit which would identify it as aberrant pancreatic tissue.—*James C. Moore, M.D.*

Paul, Lester W., and Benkendorf, Charles. Roentgen diagnosis of retrograde jejunogastric intussusception. *Radiology*, Aug., 1959, *73*, 234–237. (Address: L. W. Paul, University Hospitals, 1300 University Ave., Madison 6, Wis.)

The authors state that jejunogastric intussusception is an infrequent complication following gastroenterostomy or gastric resection. Aleman, who described 70 cases reported in the literature and 2 of his own, distinguished three types: Type I, intussusception of the afferent loop (10 per cent); Type II, intussusception of the efferent loop (70 per cent);

and Type III, combined intussusception of both loops (16 per cent).

There are two distinct clinical forms: (1) the acute and (2) the chronic.

(1) In the *acute* form the onset is abrupt with severe colicky abdominal pain which is followed by the vomiting first of food, then of bile, and finally of blood. The patient is in moderate to severe shock.

The roentgen findings consist of a filling defect, which is rounded or sausage-shaped and is situated in the dependent portion of the stomach; and thin, parallel, curved lines of barium over the surface of the mass, representing the stretched transverse mucosal folds of the jejunum. This latter feature is pathognomonic. When the intussusception occurs through a gastroenterostomy, the barium may flow through the stomach into the afferent loop when the pylorus is patent. The afferent loop may be completely obstructed, or the efferent loop may be obstructed adjacent to the gastroenterostomy. The stomach may not be dilated.

Without surgical intervention to reduce the intussusception or removal of an associated gangrenous jejunum the mortality is 100 per cent. Surgery within forty-eight hours reduces the mortality to 10 per cent; if operation is delayed more than this length of time, the mortality rises to 50 per cent.

The authors report 1 case of the acute clinical form classified as Type II intussusception of the efferent loop and present the roentgen findings in this case.

(2) In the *chronic* recurrent type of intussusception there is no characteristic clinical pattern. In most cases there is a history of epigastric pain, nausea, and vomiting occurring at irregular intervals following gastroenterostomy. Acute obstruction is more likely to occur in these patients.

The roentgen signs differ from those of acute intussusception in that the intussuscepted mass is smaller and it may be possible to demonstrate, during fluoroscopy and manual palpation of the abdomen, its spontaneous reduction or occurrence. The spiral folds of the jejunal mucosa over the surface of the mass are highly significant. There may be no abnormality noted on the roentgen examination unless the intussusception happens to occur during the examination. Examination performed when the symptoms are present is important in demonstrating this disease.—*W. M. McBride, M.D.*

Soteropoulos, C., Berkmen, Y., and Gilmore, John H. Jejunogastric intussusception. *Radiology*, Aug., 1959, *73*, 238–240. (Address: C. Soteropoulos, Illinois Masonic Hospital, 836 Wellington Ave., Chicago 14, Ill.)

The authors report a case of jejunogastric intussusception with unusual associated findings. The patient, a fifty-five year old male, had previously undergone a right radical mastectomy for carcinoma of the breast and had received radiation therapy for carcinoma of the larynx with metastases to the left lung. After admission to the hospital because of epigastric pain and vomiting, an upper gastrointestinal study was done which revealed what appeared to be a bezoar in the stomach. At laparotomy a mass of undigested string beans was removed from the stomach and an antecolic gastrojejunostomy was performed. The postoperative course was characterized by vomiting and abdominal distention. Approximately two weeks after the original surgery, a repeat upper gastrointestinal study was done which revealed a greater curvature filling defect in the region of the anastomosis. The outline and spiral appearance of the defect along with gastric retention suggested jejunogastric intussusception. At operation the patient was found to have the unusual combination of intussusception of the efferent loop and volvulus of this loop with a part of the omentum interposed. The obstruction was relieved and the patient made an uneventful recovery.

Jejunogastric intussusception is a rare complication of gastroenterostomy. The clinical findings are not characteristic and may vary from acute symptoms to chronic recurrent complaints. A definitive diagnosis requires a roentgen study with barium which will show obstruction of the stoma with a filling defect in the lumen of the stomach. As the barium outlines the intussusceptum, the jejunal folds may appear as parallel curves, a fan-shaped pattern, or as spirals. The afferent loop may fill through the stoma or by way of the duodenum in cases of simple gastroenterostomy. The authors suggest barium examination during a symptomatic period because of the possibility of spontaneous reduction in some cases.—*Edward B. Best, M.D.*

Lenz, H. Die Peristaltik des Dünndarms im Röntgen-Kinobild. (Peristalsis of the small intestine on the cineroentgenogram.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1959, *91*, 287–298. (From: Medizinische Universitäts-Poliklinik, Bonn, Germany.)

The peristalsis of the small intestine was studied by cineroentgenography having a frequency of 16 exposures per second. The peristaltic process of the small intestine occurs in two phases. The first, in preparation for emptying of the bowel, is a slowly perfected change in the tone of the musculature; the second phase is characterized by the onset of emptying peristaltic waves.

The structure of the intestinal wall is such that, at the beginning of a peristaltic wave, demonstrable remodelling of the bowel contents is brought about by the internal pressure. The filling pressure produces a stretching of the ring musculature and at the same time forces the longitudinal musculature

over the dilated portion to a contraction. The result is a bellied widening of the bowel. Through the pull of the contracted longitudinal muscle, the ring muscle segments are placed transversely to the long axis and form a narrowing. Antiperistaltic waves could not be demonstrated.—*Samuel Richman, M.D.*

Wildhack, R., and Bohlig, H. Heterotope Biligrafinausscheidung und Leber Funktion. (Heterotopic biligrafin excretion and liver function.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1959, *91*, 622–629. (From: Innere Abteilung des Städt. Krankenhauses, Lüdenscheid, Germany.)

The authors reviewed 231 intravenous biligrafin examinations done on patients with a variety of upper abdominal symptoms.

It is proved again that the use of 20 cc. of biligrafin very often results in an unsatisfactory visualization of the biliary system. An excess of contrast medium, which is accomplished by using 40 cc. of biligrafin, is necessary in order to have a satisfactory demonstration. This may lead to a corresponding "early heterotopic excretion" (usually 20–40 minutes post injection) via the kidneys, which is of no clinical significance. On the other hand, a heterotopic excretion of dye after two hours or later ("late excretion"), especially with a corresponding nonexcretion of dye via the biliary system, should be considered as due to a disturbance of hepatic function.

This fact is confirmed by corresponding bromosulphalein tests; the degree of bromosulphalein retention rises with the frequency of heterotopic "late excretion" of contrast medium via the renal system.—*Eckart Schackow, M.D.*

Chudáček, Zd., and Láhn, V. Die Bedeutung der Serumtransaminasen für die intravenöse Cholangiographie mit Biligrafin. (The importance of serum transaminases in intravenous cholangiography with biligrafin.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1959, *91*, 629–633. (Address: Zd. Chudáček, Dobrovského 11, Plzen/ČSR.)

The relationship of liver function and intravenous cholangiography is well known but, unfortunately, none of the average laboratory tests can show whether intravenous cholangiography will be successful or not in many liver diseases. The authors feel that such a prediction would be of importance, especially in two groups of patients: (1) nonicteric patients with tumor disease of the liver or gallbladder with liver metastases; and (2) nonicteric patients who had been icteric mostly because of stone obstruction.

An attempt was made to find out whether the determination of serum glutamic acid oxaloacetic acid transaminase (SGO-T) offers a possibility for predicting a positive or negative intravenous cholangiogram in these two groups of patients.

The authors' method was as follows: If cholangiography was negative after using 20 ml. biligrafin, 3, 6, and 24 hour follow-up roentgenograms were taken in order to find out whether the contrast medium could be seen in the intestinal tract. Determination of SGO-T and serum glutamic acid pyruvate transaminase (SGP-T) was done colorimetrically. There were 42 patients in the first group and 42 patients in the second group. The authors tried to exclude all those patients who may have had an elevation of the SGO-T caused by other diseases.

It was shown that intravenous cholangiography was unsuccessful in both groups, if the SGO-T level was above 2.3 μmol. Furthermore, the SGO-T test was quite significant in the first group for predicting a negative or positive intravenous cholangiography. For this purpose it was superior to corresponding serum bilirubin determinations. Of the 42 patients in this group, 12 had SGO-T levels above 2.3 μmol. and intravenous cholangiography was negative; 30 patients showed SGO-T levels below 2.3 μmol. and 27 of these had positive roentgenographic findings of the biliary system. In the second group, both tests were of equal significance in predicting the outcome of intravenous cholangiography.—*Eckart Schackow, M.D.*

Gynecology and Obstetrics

Disertori, A., and Ferrari, P. L. La colpografia dell'età pediatrica. (Colpography in the pediatric age group.) *Radiol. med.*, Nov., 1959, *45*, 1070–1077. (From: Clinica Pediatrica and Reparto Radiologico dell'Univ. di Padova, Padova, Italy.)

The authors used the following technique for the roentgenographic examination of the vagina of 50 patients whose ages ranged from seven days to twelve years.

A thin rubber catheter is introduced into the vagina, while the patient lies in the supine position. Under fluoroscopic observation, from 1.5 to 5 cc. (depending on the age of the child) of lipiodol sulfanilamide Guerbet is introduced. Roentgenograms of the lower abdomen are taken in the anteroposterior, posteroanterior and lateral views with the patient prone and supine.

The roentgenograms in the posteroanterior projection show a roundish filling defect, convex superiorly, and corresponding to the outline of the cervix. In the transabdominal roentgenograms, with the patient either supine or prone, the vagina is fully opacified and has a triangular appearance in early childhood, while it becomes cylindric and turned upward from its distal end in later years.

The authors are of the opinion that this procedure is indicated in cases of congenital abnormalities of the vagina or the uterus, in the diagnosis of neoplasms, in the demonstration of radiolucent foreign bodies and in the study of the development of the sexual organs.—*A. F. Govoni, M.D.*

MACARINI, N., SCURSATONE, M., and ZINICOLA, N. L'arteriografia pelvica nello studio della patologia ginecologica. (Pelvic arteriography in the study of gynecologic pathology. *Radiol. med.*, Nov., 1959, *45*, 1041–1069. (From: Istituto di Radiologia and Istituto di Patologia Chirurgica dell'Università di Genova, Genova, Italy.)

The importance and usefulness of arteriography of the iliac arteries and its branches in the study of gynecologic and obstetric conditions was demonstrated by the studies conducted by Borell and his associates (*Acta radiol.*, 1952, *38*, 247; 1953, *40*, 561; 1954, *42*, 253). Several techniques are recommended; the authors have adopted the one suggested by Castellanos and Pereiras (AM. J. ROENTGENOL. & RAD. THERAPY, 1950, *63*, 559) and Borell *et al.*

They have found that, for a better and more complete visualization of the uterine circulation, arteriography should be performed during the menstrual period, when a greater number of vessels can be opacified and, in particular, the intramural ones.

Eleven cases with different gynecologic conditions are presented and the roentgenographic findings are discussed.

In uterine fibroma (4 cases), when the mass is intramural and located in the body, a displacement of the marginal artery, which shows a concavity in its medial aspect, is noted. When the lesion is in the fundus, the marginal segment of the uterine artery appears to be displaced laterally on both sides. In the case of a subserous fibroma, the displacement of the uterine artery is not always evident and usually there is an increase in the caliber of the artery. Several newly formed vessels are seen around and in the mass, in particular during the capillary stage. It should be emphasized, however, that not all these findings may be present in the same individual.

In benign cystic ovarian tumors (2 cases) the only roentgenographic sign is the displacement of the uterine, ovarian, mesenteric and adnexial arteries and, if the mass has a large diameter, also of the common, external and internal iliac arteries. The mass is not opacified and there is a rich arterial bed at the periphery. In the case of malignant cystic neoplasms of the ovary (2 cases) there is early opacification of the mass, with evidence of vascular pools in the mass itself and an early venous stage; there is also a typical change in the adnexal artery, which is increased in diameter and ends abruptly in numerous, irregular, small branches.

In malignant neoplasms of the uterus (3 cases), the whole vascular bed is completely disrupted and is replaced by an irregular mass of vessels which are of various calibers and follow very irregular courses. A few vessels end in lacunar formations where the contrast medium remains for quite a while. There is also a very early venous return, since the normal circulatory system has been replaced by a vascular network of embryonic type with arteriovenous shunts.

Contraindications for this angiographic examination are the presence of hepatic or renal diseases, advanced tuberculosis of the lung, hyperthyroidism, cardiac failure and hypersensitivity to iodine. In 1,000 cases the authors did not observe any secondary accident.

The dose received by the gonads during the procedure (4 roentgenograms are usually taken) is about 2 r, similar to that received by the gonads in roentgen pelvimetry.—*A. F. Govoni, M.D.*

GENITOURINARY SYSTEM

CHYNN, KUO YORK, and EVANS, JOHN A. Nephrotomography in the differentiation of renal cyst from neoplasm: a review of 500 cases. *J. Urol.*, Jan., 1960, *83*, 21–24. (From: Department of Radiology, New York Hospital-Cornell Medical Center, New York, N. Y.)

The method used by the authors has been carried out on 500 consecutive patients during a five year period. Indications for the procedure are found in those patients in whom it is important to differentiate renal malignancy from benign renal cysts. The procedure is of considerable value in those patients who are poor risks for surgery, and in whom differentiation cannot be made on routine urographic studies.

The principles which underlie the procedure consist of opacification of the kidneys followed by routine roentgenograms and tomograms. Renal tumors will show increased density with abnormal blood vessels which can be distinguished from the surrounding normal renal parenchyma. Necrosis in tumor will produce blotchy, poorly defined areas of radiolucency. Cystic lesions show one or more sharply defined radiolucent areas, which are well demarcated from surrounding functioning renal parenchyma.

Following preliminary roentgenograms of the abdomen, tomograms are taken to determine exposure factors and optimum levels for tomography. A No. 12 Robb-Steinberg needle is placed in the antecubital vein and its position is checked with normal saline solution. The circulation time is then determined using 2.5 cc. of 50 per cent decholin mixed with 15 cc. of normal saline. After this is established by having the patient respond the instant the bitter taste is noted, 30 cc. of 50 per cent hypaque is injected. Another syringe containing 50 cc. of 90 per cent lukewarm hypaque is then placed on the in-

dwelling intravenous needle and this solution is injected as rapidly as possible. The time for injection should not be more than one and one half seconds. This will opacify the aorta and renal arteries on the survey roentgenogram made at the appropriate time, as indicated by the circulation time. Tomograms are made immediately following the abdominal roentgenogram. These are then developed and studied and, if necessary, a repeat second injection may be done.

There have been no serious side effects. The patients may have a sensation of heat throughout the body, but this does not last more than a few seconds. Vomiting occurs in less than 5 per cent and the incidence of phlebitis is no greater than that seen in patients undergoing intravenous urography.

This method yields a high degree of diagnostic accuracy for the differentiation between renal cyst and carcinoma. The percentage of error is approximately 5 per cent in all cases studied.—*George W. Chamberlin, M.D.*

Hoffman, Howard A., and de Carvalho, Miguel Paes. Retrograde pyelography and use of contrast mediums containing neomycin. *J.A.M.A.*, Jan. 16, 1960, *172*, 236. (Address: H. A. Hoffman, 60 Eighth St., New Bedford, Mass.)

Recent experiments have been performed with products containing determined percentages of antibiotics in an attempt to avoid reactions that occur after retrograde pyelography. However, the authors suggest that it might be of greater benefit to reconsider basic principles of technique than to rely on covering up the damage with antibiotics. It is their impression that there is no difference in the result (*i.e.*, reactions after retrograde pyelography) whether the gravity instillation method with the patient in the Trendelenburg position is employed or a slow injection of medium is carried out with the patient in the horizontal position. A sensation of fullness in the flank may be considered normal, but the examiner should not have to depend on a colic to realize that sufficient medium has been instilled. Overdistention of the pelvis is probably responsible in most patients for the immediate reaction of pain in the kidney area.

Various textbooks of urology suggest unduly large quantities of contrast medium as proper for instillation, such as 8 to 15 cc., 7.5 to 20 cc., etc. This is most unfortunate, since pursuing this practice invariably will lead, in a renal pelvis of normal capacity, to the "overdistention reaction." The authors state that in the vast majority of their adult patients, in whom hydronephrosis or pyelectasis was not previously suspected, 2.5 to 3.0 cc. of contrast medium was adequate to obtain the desired delineation of the collecting system. The usual adult pelvis is not prepared to accept more than 3 cc; in children 2 cc. should be maximal, and in infants 1 cc.

Controlled studies, carried out by the authors in two consecutive series of 100 patients each, showed no clinical difference between the simple contrast medium (methiodal sodium) and the one containing 2.5 per cent of neomycin sulfate.

The authors conclude that the prophylactic or therapeutic value of the addition of neomycin sulfate to opaque media seems insufficient to justify its use at an increased cost. Reactions after retrograde pyelography would be more apt to be prevented if textbooks accurately stated the anatomic capacity of the renal pelvis and if this were heeded by the cystoscopist.—*D. S. Kellogg, M.D.*

Veiga-Pires, J. A. Pyelo-ureteritis cystica; a report of two cases. *J. Fac. Radiologists*, Oct., 1959, *10*, 207–212. (From: Royal Northern Hospital, Holloway, London, England.)

The author describes 2 cases, 1 of pyelitis cystica and 1 of pyelo-ureteritis cystica. The condition is characterized by small cysts which may vary in color from hemorrhagic pink to gray and which contain a more or less viscous fluid. The lesions are palpable, giving a shot-like feel to the ureter or pelvis. It is believed that the condition has its origin in an underlying inflammatory process but lymphatic stasis may be a contributing factor.

The most common complication of pyelo-ureteritis cystica, particularly if it involves the upper ureter, pelvis, or pyelo-ureteric junction, is obstruction producing hydronephrosis, with progressive destruction of the renal parenchyma. There is a potential danger of malignant change leading to adenocarcinoma, especially in the case of lesions in the bladder.

There is a history suggestive of a chronic urinary infection with loin pain. Dysuria and frequency are quite common. Pyuria and hematuria are almost constant findings. The signs can be minimal even when the condition is advanced. Cystoscopy may reveal small cysts, situated mainly in the region of the trigone, with a background of inflammatory edema, or may show a normal bladder. Over 90 cases have been found on searching the literature.

On excretion pyelography, renal function will vary with the extent of the disease, the degree of mechanical obstruction and the severity of the infection. Pelvic compression should not be used throughout the examination, since it might obscure small alterations of outline or any structures protruding into the lumen. Retrograde pyelography is the investigation of choice.

The cysts, if widely spaced, produce punched out defects in the lumen. If they form clusters and are relatively small, they give a ragged appearance, particularly in the ureter. More or less gross hydronephrosis and hydroureter may develop. The roentgenographic picture may remain unchanged for years.

When the condition is advanced, the ureter may appear elongated and sinuous with zones of ring-narrowing alternating with dilatations due to fibrotic changes.

The most common condition simulating ureteritis cystica is the presence of air bubbles in the renal tract at any level. Other conditions which may produce difficulties in differential diagnosis are non-opaque calculi, blood clots, and tuberculous filling defects in the ureter, and papillomata of the pelvis or the ureter.—*Samuel G. Henderson, M.D.*

Mitsuya, Hideo; Asai, Jun; Suyama, Keiji; Ushida, Takao; and Hosoe, Kenzo. Application of x-ray cinematography in urology: I. Mechanism of ejaculation. *J. Urol.*, Jan., 1960, *83*, 86–92. (From: Department of Urology and Department of Radiology, Nagoya University School of Medicine, Nagoya, Japan.)

Following the injection of radiopaque material into the lumen of the vas deferens, roentgen-ray studies and cinematography using the Philips image intensifier were carried out on 16 subjects who were free of any urologic complaints at the time of the examination.

Detailed studies show the physiology and anatomic variations associated with the vas, ampulla, and seminal vesicles during the various phases of sexual excitement and ejaculation. These findings are coordinated with other studies of the physical and chemical nature of the ejaculatory fluid.—*George W. Chamberlin, M.D.*

Nicolai, Charles H., and Spjut, Harlan J. Primary osteogenic sarcoma of the bladder. *J. Urol.*, Oct., 1959, *82*, 497–499. (Address: C. H. Nicolai, 4960 Audubon Ave., St. Louis 10, Mo.)

This is a single case report which presents a sixty-six year old patient who was found to have a large pedunculated tumor involving the bladder wall. The cystogram showed a filling defect, 5 cm. in diameter, in the mid-portion of the bladder without evidence of calcification or ossification within it. This area, including a section of the bladder wall, was removed. The tumor was firm, glistening, and yellowish-white in color. It invaded the muscular layers of the bladder, and showed considerable necrosis and some areas of osteoid tissue and cartilage. Calcified bone was not seen. A few minute segments of epithelial cells were present and there were some regions in which the cells tended to be spindle shaped with a myxomatous background.

Primary osteogenic sarcoma of the urinary bladder is a rare form of disease. It has previously been reported in 11 patients. Most of these patients demonstrated extensive infiltration of the bladder and, in 4, there was evidence of distant metastases. The histogenesis is not known, but it has been suggested that metaplasia from epithelium to bone and cartilage may explain this unusual tumor.

Postmortem examination in the authors' case showed that the bladder tumor had been cleanly removed, suggesting that it is possible by surgical methods to eradicate such lesions.—*George W. Chamberlin, M.D.*